Chapter 3

Diseases of the Bovine Gastrointestinal Tract

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I. BOVINE FORESTOMACH AND ABOMASUM

A. Indigestion. The primary clinical signs for this condition are anorexia and ruminal changes characterized by hypomotility or occasionally hypermotility.

1. Simple indigestion
   a. Patient profile and history
      (1) Simple indigestion is a common disease in dairy cattle and less common in feedlot cattle and other ruminants. The condition is sporadic, usually affecting individual cows, but groups can be affected. This type of indigestion occurs more frequently in older cows, greedy eaters, or cows in advanced pregnancy.
      (2) There may be reported changes in the feeding program (i.e., quality, quantity, frequency) or other management changes. The owner might report that the cow is off feed and down in milk production. Feed refusal may have been progressive in that grain may have been refused first, followed by silage and hay.
   b. Clinical findings. The cow may be partially to completely anorexic. Vital signs [temperature, pulse, and respiration (TPR)] are normal to slightly elevated. The animal has a normal to mildly depressed attitude. Rumen motility may be normal but usually is slightly decreased in frequency and vigor. Feces may be normal in consistency or firm, and fecal output usually is reduced. Occasionally, the rumen is hypermotile, resulting in feces that are looser than normal.
   c. Etiology and pathogenesis
      (1) The condition is caused by a change in rumen fermentation resulting from a shift in feed quality, quantity, or presentation. Some predisposing factors in the development of the condition include:
         (a) Sudden changes of feed
         (b) Poor feed quality (e.g., moldy, spoiled)
         (c) Animal fatigue or stress (e.g., shipping)
         (d) Prolonged antibiotic therapy
         (e) Insufficient water
      (2) Any of the predisposing factors might change the ruminal environment necessary for fermentation and microbial degradation of feedstuffs. The rumen environment is composed of a mixture of proteins, carbohydrates, and fluid. Bacteria and protozoa act on these substances within an environment with a pH and temperature that is regulated by secretion and motility.
   d. Diagnostic plan. The history, lack of specific findings other than minor gastrointestinal changes, and a knowledge of the farm husbandry usually is sufficient to make the diagnosis. It is often a diagnosis by exclusion of other diseases, and the animal's recovery within 24–36 hours confirms the diagnosis.
   e. Laboratory tests. Laboratory tests ordinarily are not requested because all values usually appear normal. Occasionally, the cow may exhibit a "stress" leukogram. The rumen pH may be slightly alkaline (6–7) and show somewhat decreased protozoal activity. A mild metabolic alkalosis also may be present
   f. Differential diagnoses. Other conditions to be considered would have clinical signs in addition to mild indigestion. These conditions include:
      (1) Traumatic reticuloperitonitis
      (2) Abomasal displacements
      (3) Vagal indigestion
      (4) Primary ketosis
      (5) Lactic acidosis
   g. Therapeutic plan

(1) Often, little if any therapy is necessary. Many owners diagnose the condition themselves, treating the animal with over-the-counter rumenatorics or providing good quality feed and water.
(2) Rumenatorics may contain nux vomica, capsicum, ginger, and tartar emetic. There is little need for rumen stimulants such as neostigmine. Cathartics, such as magnesium hydroxide (MgOH) or magnesium oxide (MgO), should be avoided because they exacerbate the mild metabolic alkalosis that may already be present. MgOH or MgO would be indicated if the rumen pH is below 5.5. If a primary cathartic is indicated, a better choice would be magnesium sulfate (500–1000 g orally).
(3) The best treatment is the provision of quality, long-stemmed hay and fresh water. The owner should monitor the animal's response and request a revisit 24 hours later if there is no improvement.
(4) If the animal does not respond within 24 hours, a clinical examination should be performed again. If the diagnosis is still simple indigestion, consider a rumen transfaunation with 1–2 L of fresh rumen juice. Before performing the transfaunation, clinicians may wish to adjust the rumen pH to physiological levels by administering alkalinizing agents (in the case of an acid rumen) or acidifying agents (vinegar 1–2 L orally) if the rumen pH is more than 7.

h. Prevention. For prevention of this condition, the client should be instructed to minimize sudden dietary changes and avoid damaged, spoiled, or contaminated feed.

2. **Lactic** acidosis (**ruminal** acidosis, acute grain overload, acute **rumen** impaction, **rumen** overload, **D-lactic** acidosis, grain engorgement, toxic indigestion, acid indigestion)

a. Patient profile and history
(1) Any ruminant is susceptible to lactic acidosis. Dairy and beef cattle seem to be most commonly affected possibly because of their representative numbers or husbandry and intensive production practices.
(2) Patient history includes access to highly fermentable feed. The offending substances are highly soluble carbohydrates such as finely ground grains (e.g., wheat, barley, corn), apples, pears, potatoes, bakery products, beets, whey, and brewers grain.
(3) There may be a history of poor feeding management or inconsistent concentrate feeding.

b. Clinical findings are variable and depend on the amount of feed consumed, feed composition, feed particle size, and previous adaptation of the animal to the ration. Clinical syndromes may vary from acute and severe to mild and similar to simple indigestion. A chronic form of lactic acidosis may also occur.
(1) Acute severe cases
(a) Clinical signs appear 12–36 hours after the feed is consumed. Symptoms include anorexia, lethargy, depression, muscle tremors, and ataxia.
(b) The animal is dehydrated (8%–12%), resulting in loss of skin turgor and a dull, sunken eye. There is severe ruminal distention, rumen stasis, and fluid splashy rumen contents. The animal may exhibit bruxism and grunting.
(c) Body temperature initially increases and then falls. There is tachycardia and a rapid, shallow respiratory pattern. Dairy cattle have a severe drop in milk production. The animal may appear blind, with a sluggish palpebral and eye preservation reflex.
(d) It may take 24 hours for diarrhea to develop, but the feces then appear foamy and liquid with an acid smell and a yellow-brown or grey color. The feces may contain undigested feed material (e.g., grain).
(e) The animal continues to deteriorate, becoming recumbent, comatose, and finally dying.
(2) Subacute cases. The pattern of clinical findings is similar to the acute cases but less intense.
(3) Chronic cases
(a) Repeated episodes of subacute rumen acidosis may be associated with herd problems of subclinical disease (e.g., laminitis, low-fat milk syndrome, liver abscesses, chronic rumenitis, chronic indigestion).
(b) Individual episodes of indigestion appear mild and similar to simple indigestion or subacute indigestion. Animals appear bright and alert but with transient anorexia and decreased effective rumen motility. The rumen may be slightly distended. Feces are grey and porridge-like. Dairy cattle experience decreased milk production.

c. Etiology and pathogenesis
(1) Normally, there is a balance of **cellulolytic** and carbohydrate-using **bacteria** within the rumen. The ingestion of excess carbohydrates or sugars promotes growth of lactic-acid-producing and -using bacteria within the rumen. Although lactate is used rapidly, the amount that accumulates plus the rapid fermentation of carbohydrates and the accumulation of volatile fatty acids (VFAs) drop the rumen pH. This decrease in pH kills rumen protozoa and microbes, including the initial lactate users. These organisms are replaced first by Streptococcus bovis and then by lactobacilli and gram-positive rods. The pH continues to fall with the further lactobacilli production of D- and L-lactic acid.
(2) Finely ground feeds increase the surface area exposed to bacteria for fermentation. These feeds also decrease the amount of saliva secreted by the animal, which lessens the amount of buffer flowing into the rumen. Although both D- and L-lactic acid are absorbed through the rumen wall, only L-lactic acid can be metabolized by the ruminant, leaving the D-lactic acid to be eliminated. Therefore, D-lactic acid builds up and creates the systemic acidosis.
(3) Feed fermentation, decomposition of the feed into very fine particles, and the lactic acid all increase the **rumen osmolality.** The accumulation of VFAs produce rumen stasis. Extracellular water flows into the rumen, producing rumen distention, diarrhea, and dehydration. These conditions lead to hypovolemia, circulatory collapse, metabolic acidosis, and death.
(4) If the animal survives the initial bout of lactic acidosis, the high rumen acidity, hypertonicity, and corrosive nature of lactic acid produce a chemical **rumenitis.** This condition allows rumen-associated bacteria and fungi to invade the rumen wall and hepatic portal system.
(5) Sequelae include rumen wall necrosis, hepatic abscess, and peritonitis. The release of toxins may produce laminitis, abomasal disorders, and cardiac, renal, and hepatic damage.

d. Diagnostic plan. The diagnosis often is determined based on clinical findings supported by a history of overeating or sudden dietary change. A sample of rumen fluid should be retrieved and analyzed.

e. Laboratory tests
(1) Laboratory support is rarely **necessary** in acute cases where there is a reliable history. The most valuable laboratory aid is an evaluation of rumen content for subjective findings, pH, and protozoal activity.
(2) The **rumen** fluid retrieved by orogastric intubation is milky grey and watery with an acid smell. The pH is variable, depending on time and diet, but it is diagnostic if less than 5.0. A wet mount shows no live protozoa. A Cram stain shows Streptococci with a predominant population of gram-positive rods and other mixed, mainly gram-negative, morphologic forms.
(3) Hematologic work-up. A leukogram reveals a degenerative left shift. Hemoconcentration is evident on a packed cell volume (PCV). A chemistry panel shows an increase in blood urea nitrogen (BUN) and creatinine, hypocalcemia, hypomagnesemia, and hyperglycemia.
(4) Urinalysis shows decreased volume, increased specific gravity, aciduria, and glucosuria (often a diagnostic indicator in sheep). If available, a blood gas analysis confirms a metabolic acidosis.

f. Differential diagnoses. Lactic acidosis may appear similar to many other septic or toxic conditions including:
(1) Septic mastitis or metritis
(2) Acute diffuse peritonitis
(3) Parturient paresis

(4) Poisoning by lead, salt, arsenic, or nitrate
(5) Enterotoxemia

g. Therapeutic plan. It is often difficult to determine therapeutic intensity with acute lactic acidosis, particularly early on when the full range of clinical signs has not been manifested. Some guidelines for treatment follow.
(1) There has been access to carbohydrates but the animal(s) has not shown clinical signs.
(a) Prevent further access to feed, and offer free choice, good quality hay.
(b) Exercise the animal(s) hourly for 12–24 hours to encourage movement of ingesta through the digestive tract.
(c) Administer 1 g/kg of magnesium oxide, magnesium hydroxide, or sodium bicarbonate orally.
(2) The animals show anorexia and depression within 6–8 hours of feed consumption.
(a) Surgery. Perform a rumenotomy if the value of the animal warrants the procedure and if the case is still a good surgical risk. Tachycardia (greater than 140 beats/minute), dehydration, a subnormal body temperature, and severe depression indicate a poor prognosis for recovery. Couple surgery with fluid therapy for metabolic acidosis (e.g., Ringer's Lactate with sodium bicarbonate), and supply a rumen transfaunation. Other supportive therapies include calcium, thiamine, and nonsteroidal anti-inflammatory drugs (NSAIDs).
(b) Recommend that the animal be slaughtered if economics do not dictate surgery and the animal will pass an antemortem and post-slaughter inspection.
(c) Other treatments. Other less predictable or efficacious treatments include rumen lavage via rumen trocar, systemic or oral antibiotics to limit ruminal bacterial growth, and oral iodine-based disinfectants to kill rumen microbes. Because of the potential for a large number of animals to develop lactic acidosis simultaneously, the use of a large-diameter stomach tube (Kingman tube) to lavage the rumen may be both economic and beneficial.
(3) Animals with chronic lactic acidosis. Individual animal treatment is often unsuccessful and usually not warranted. This is most commonly reflective of a herd problem and should be addressed in a preventive manner.

h. Prevention
(1) Management strategies. Recommendations should revolve around ration and feeding management. Keep feed quality and practices consistent. Avoid abrupt changes and gradually adapt the rumen to concentrates. When introducing new concentrate or carbohydrate forms, prefeed animals with hay. Prevent accidental access to feeds by maintaining good animal holding facilities.
(2) Feed content. Maintain a minimum crude fibre content of 14% of total digestible nutrients (TDN) for fattening cattle and 18%–22% for dairy cattle.

3. Vagal indigestion (chronic or vagus indigestion, Hoflund's syndrome). Vagal indigestion refers to a group of conditions that cause forestomach outflow problems. One possible cause of these problems is vagal nerve dysfunction, although this dysfunction has not been proven to be necessary or sufficient to cause the condition. The classifications of forestomach disturbances encompassed by vagal indigestion differ from author to author. Therefore, this section discusses the syndrome as a whole and delineates subtypes as necessary for diagnosis, therapy, and prognosis.

a. Patient profile and history. Although any ruminant may be affected, this type of indigestion is most common in adult dairy cattle. History may include mild but repeated bouts of transient indigestion with signs of anorexia, decreased milk production, mild bloat, weight loss, abdominal distention, and decreased amounts of manure. An episode of traumatic reticuloperitonitis (TRP) may be reported as an historical event.

b. Clinical findings. A distended abdomen is often a cardinal sign. The distention may be bilateral with gas distending the upper left flank, while fluid distends the ventral right quadrant. The rumen is often hypermotile with frequent but weak contractions. The animal is often in poor condition. Vital signs are usually normal but occasionally, a bradycardia is evident (40–60 bpm).

c. Etiology and pathogenesis
(1) Vagal indigestion is most often described as a sequela to traumatic reticuloperitonitis, although the syndrome is also associated with tumors (e.g., lymphosarcoma of the abomasum) and abomasal torsion.
(2) If adhesive lesions around the reticulum either interfere with vagal nerve function or the mechanics of reticular movement, then reticuloruminal cyclical activity and eructation waves are compromised, resulting in ruminal gas retention or free gas bloat. In this case, there are no other abnormalities, and the animal will improve transiently if the gas is removed via orogastric tube and recover fully if a rumen fistula is installed.
(3) If failure of omasal transport occurs, ingesta from the ruminoreticulum is not properly conveyed to the abomasum, and long (2–4 cm) fiber may be found in the manure. Fluid builds up in the rumen, and although acid–base status remains normal, the animal becomes mildly dehydrated. The prognosis for a return to function is guarded. The lesion causing the condition is either a functional or physical obstruction of omasal outflow. Examples of physical obstructions include ingested foreign bodies (baling twine, plastic bags, placenta) or space-occupying lesions (tumor).
(4) Abomasal impaction may be another manifestation of the syndrome and results from obstruction at the pylorus with conditions such as lymphosarcoma. This impaction also may occur secondary to abomasal torsions, where it is postulated that abomasal stretching and visceral compromise impairs gastric motility. The abomasum becomes distended with hard ingesta. A hypochloremic, metabolic alkalosis results due to reflux and pooling of gastric chloride ion in the rumen. This "chloride trap" makes it unavailable for reabsorption in the proximal small intestine.
(5) Primary abomasal impaction may occur in beef cattle on a coarse roughage diet with limited access to water and in calves suffering from abomasal trichobezoars or phytobezoars. These conditions are not usually considered as part of the vagal indigestion syndrome.
(6) The final manifestation of the syndrome is chronic indigestion associated with advanced pregnancy. The gravid uterus may occupy enough abdominal space that it interferes with forestomach outflow. It may also exacerbate reticular adhesions or abomasal outflow restrictions, resulting in ruminal distention. The prognosis with this condition is often fair to good following parturition.

d. Diagnostic plan
(1) The set of clinical signs, chronicity, and continued lack of response to any treatment for simple indigestion is usually sufficient to diagnose vagal indigestion. The presence of a metabolic alkalosis often implicates the abomasum in the syndrome but this is not invariable.
(2) Differentiation of the causes may depend on findings on exploratory laparotomy; however, mixed conditions do occur and lesions are not invariably present. The poor response to interventive therapy with many of these cases often makes a morphologic diagnosis unnecessary.

e. Differential diagnoses. It is often necessary to rule out simple indigestion and many other causes of decreased fecal output and abdominal distention (e.g., abomasal displacement, hydrops). Vagal indigestion is often a diagnosis of exclusion.

f. Therapeutic plan
(1) Surgical exploration is often used in valuable animals as an aid to diagnosis and therapy. In cases of free gas bloat, the creation of a rumen fistula often enables a return to ruminal function while the underlying lesion heals. Indwelling rumen trocars have been used to relieve the bloat but are less reliable than surgery. The prognosis for vagal indigestion involving omasal or abomasal dysfunction is relatively poor, so slaughter should be considered as an option in these cases.
(2) In cases where exploratory surgery has been performed, further surgical intervention depends on findings. Foreign bodies (in the abomasum or fore-

stomach) should be removed, abscesses adherent to the reticulum can be drained into the reticulum, and softening agents (e.g., mineral oil) can be instilled into the abomasum by an orogastric tube directed through the omasal–abomasal orifice intraruminally.

(3) Supportive therapies include:
- (a) Rumen transfaunation (repeated treatments may be necessary)
- (b) Supportive feeding either by rumen fistula or an indwelling nasogastric tube that has been placed into the abomasum at surgery
- (c) Intravenous fluids
- (d) Calcium salts intravenously or subcutaneously
- (e) Potassium chloride at 30–60 g orally twice a day
- (f) Exercise

g. Prevention. There is little that can be done to prevent vagal indigestion. However, if chronic reticular adhesions indicate that traumatic reticuloperitonitis may be occurring in a herd, it would be good to administer magnets to breeding-age heifers as a preventive. Other measures to prevent foreign body or feed impactions from occurring should be implemented by decreasing access to indigestible substances (e.g., rope, twine, plastics).

4. Abomasal impaction

a. Patient profile. Primary abomasal impaction occurs most commonly in beef cattle and is most often seen in pregnant cows.

b. Clinical findings. Animals present with anorexia, scant feces, variable dehydration, and moderate abdominal distention. The distended, hard abomasum may be palpated in the lower right abdomen.

c. Etiology and pathogenesis. The pathogenesis is most commonly one of excessive intake of poor quality roughage at times of increased energy needs (i.e., pregnancy, winter months). Mechanical obstruction by a foreign body or pyloric mass is another cause. Obstruction results in excessive accumulation of rumen chloride, causing a metabolic alkalosis. A progressive starvation ensues.

d. Diagnostic plan. The diagnosis is based on clinical findings. The condition appears similar to vagal indigestion but is often a herd problem. Laboratory information helps narrow the diagnostic possibilities. Exploratory surgery confirms the condition.

e. Laboratory tests show metabolic alkalosis, hypochloremia, hypokalemia, and hemoconcentration.

f. Therapeutic plan
- (1) By the time clinical signs develop, treatment usually is unrewarding. Slaughter of meat animals may provide some economic return.
- (2) Measures undertaken for selected individuals include induction of parturition, surgery, softening agents per os, and supplemental nutrition. However, rarely are these heroic measures effective.

g. Prevention. Recommendations for prevention include provision of adequate amounts of nutrition for maintenance and pregnancy, particularly in the cold winter months. Nutritional counseling may be necessary for owners trying to maintain cattle on low-input regimens. The importance of good quality drinking water should not be overlooked.

B Bloat (ruminal tympany)

1. Acute **bloat.** Acute or sudden-onset bloat can be divided into two types: frothy bloat and free gas bloat.

a. Patient profile and history. Bloat occurs more commonly in cattle than other ruminants, and certain individual cattle may be more susceptible than others.
- (1) Frothy bloat is often associated with dairy cows that recently have been turned out on new-growth, lush pastures (e.g., alfalfa, clover). Several animals may be affected. Feedlot cattle can also develop frothy bloat when more than 50% of their ration is being consumed as concentrate.
- (2) Free gas bloat is associated with a history of feeding whole or only partially chopped solid feeds (e.g., potatoes, apples, turnips).

b. Clinical findings. Animals present with severe abdominal distention and increased TPR. The distention initially is restricted to the left dorsal abdominal quadrant, but in severe cases the right flank will also distend. The rumen may be hypermotile or hypomotile. Animals have excessive salivation and are anxious. Animals will mouth-breathe, become recumbent, and quickly die.

c. Etiology and pathogenesis
- (1) Frothy bloat is associated with legume consumption.
  - (a) It is thought that the fine, thin leaf structure of certain varieties of legumes coupled with tender growth (early or late season) allows for more rapid bacterial degradation and intraruminal particle suspension. Chloroplast released from the legume leaf forms monomolecular foams that trap gas bubbles. These foams have great surface tension and are highly stable.
  - (b) The result is that small gas bubbles do not coalesce, the cardia or the forestomach cannot be cleared of this foam, and the animal is unable to eructate.
  - (c) A stable froth can also be formed in feedlot animals consuming a primarily finely ground grain diet. In this case however, a mucoprotein slime stabilizes the foam. This foam is stable at a low pH created by lactate and VFA production. Salivation is decreased because of the fine grind of the diet, which also lessens intraruminal buffering.
- (2) Free gas bloat may have a variety of causes. The most common cause is intraesophageal **obstruction** with solid objects, such as apples or potatoes. Extraesophageal masses may also cause the build up of intraruminal gases. Abscesses caused by perivascular injections, *Hypoderma lineatum* reactions, and cervical neoplasia can all constrict the esophagus. Certain postures or diseases also can produce a functional free gas bloat. Examples include milk fever and tetanus. Moderate free gas bloat also may be a finding in vagal indigestion (see I A 3).

d. Diagnostic plan. An accurate history and the passage of an orogastric tube rapidly determines whether the condition is one of abdominal distention resulting from the accumulation of ruminal free gas or froth. If the tube cannot be passed, then the likely diagnosis is free gas bloat due to esophageal obstruction. If the tube can be passed but ruminal gas is not readily forthcoming, then frothy bloat is likely. Remove the tube and look at its end for evidence of froth. A reading of the foam pH further defines the type of frothy bloat; a feedlot bloat should have a pH of less than 5.5.

e. Differential diagnoses. If the diagnosis is not one of bloat, other causes for abdominal distention to rule out include ascites, acute diffuse peritonitis, and hydrops.

f. Therapeutic plan
- (1) Acute bloat is one of the true medical emergencies in bovine practice. Animals can die rapidly and many may succumb before the owner is aware of the problem, particularly if cattle have been turned out to new pasture for an unobserved length of time. In the case of free gas bloat, lethal ruminal pressures also build up very rapidly.
- (2) With frothy bloat, pass an orogastric tube and administer an oil to reduce the surface tension of the foam and allow the gas bubbles to coalesce. Either mineral oil at 1 L/100 kg orally or dioctyl sodium sulfosuccinate (DSS) in peanut oil at 17–66 mg/kg orally may be used (150–600 ml/450 kg animal). The treatment should be satisfactory and sufficient if the animal is still standing and not showing evidence of respiratory or cardiac failure. If the animal is in a deteriorating clinical condition, an emergency **rumenotomy** is warranted.
- (3) With acute free gas bloat due to an intraesophageal mass, the orogastric tube may force the obstruction into the reticulum. Should this not occur, trocarization of the rumen and either an indwelling trocar or fistula is placed to allow continued escape of gas while the mass softens and hopefully moves along. Post-surgical penicillin (22,000 IU/kg twice daily intramuscularly) should be used. Many masses block the esophagus just posterior to the pharynx. These can often be retrieved manually or with the aid of a wire loop to snare the object.

g. Prognosis

(1) The prognosis for most cases of **frothy bloat** is favorable if intervention is rapid. A simple indigestion may occur secondary to treatment, and the animal should be fed good quality hay as a major component of its diet for 1–2 days. Animals that have undergone emergency rumenotomies may develop cellulitis or peritonitis, but these occurrences are infrequent.

(2) The prognosis for **acute free gas bloat** is excellent if the offending object can be removed. If the object must be left in place to be swallowed, then **complications** include sequelae to trocarization or rumen fistulation as well as secondary esophageal stricture development.

h. Prevention

(1) The prevention of **frothy bloat** may take many forms and several combinations including:

(a) If legumes are fed, select cultivars of bloat-producing forages that are less likely to ferment quickly.

(b) Feed dry roughage to animals before turning them out on legume pastures.

(c) Allow only 20 minutes initially of grazing on lush legume pastures.

(d) Feed antifoaming materials (poloxalene at 1–2 g/50 kg once daily; ionophore drugs such as monensin at 1 mg/kg once daily or lasalocid at 1.32 mg/kg once daily in nonlactating cattle).

(e) Provide for slower introduction or less heavy feeding of concentrates if feedlot bloat becomes a problem.

(2) The prevention of **free gas bloat due to esophageal choke** becomes a matter of cutting up feed objects in smaller sizes, slowing down greedy eaters by feeding hay first upon initial introduction, or introduction of the material gradually.

2. Chronic free gas bloat (see I A 3)

C. Hyperresonance

1. Left displacement of the abomasum (abomasal displacement, twisted stomach)

a. Patient profile and history

(1) Left displacement of the abomasum (LDA) is most common in **middle-aged, high producing dairy cows** but may be seen in other situations and classes of cattle. This condition is rare in other ruminants but may occur in ruminating calves. It is a sporadic disease of individual animals, but the prevalence may be relatively high in some herds.

(2) Cattle are often in **early lactation** on a **high concentrate, low roughage diet.** Total mixed rations with short fibre lengths and silage-based rations seem to predispose cattle to the condition. There is often a concurrent disease, such as metritis, mastitis, or hypocalcemia.

b. Clinical findings

(1) **Vital signs** are normal to slightly elevated unless there is concurrent disease.

(2) **Appearance.** The animal may have a slab-sided appearance or the last two ribs on the left may be sprung with a hollow left paralumbar fossa. In some cases, there may be a slight filling of the left paralumbar fossa directly behind the last rib.

(3) **Feces.** There are decreased amounts of pasty manure or small amounts of diarrhea.

(4) **Rumen contractions** are normal to slightly decreased. There may be tinkling sounds heard while auscultating the rumen. The animal exhibits total to moderate anorexia and may be ketotic. A **"ping"** is evident on simultaneous auscultation and percussion of the abdomen usually along a line drawn from the left tuber coxae to the left elbow.

(5) On **palpation** of the left paralumbar fossa, the rumen is not palpable, but the dilated abomasum occasionally may be felt. Rectal examination may be relatively normal except for a smaller than normal, medially deviated rumen.

(6) **Calves** with LDA exhibit quite an extensive left flank distention, with gas and fluid sounds on auscultation.

c. Etiology and pathogenesis

(1) The **abomasum** is firmly attached at its cranial end to the omasum, but the fundus and pylorus are relatively freely moveable, being held only by the greater and lesser omentum. The abomasum continually generates gases (CO_2, methane, nitrogen) and secretes HCl and enzymes, which are passed into the duodenum by abomasal contractions (1–2/min).

(2) **High concentrate, low roughage diets** are thought to cause increased VFA production in the rumen, leading to an increase in VFAs in the abomasum, increased gas production in the rumen, and accumulation in the abomasum. This diet also may cause decreased stimulation to rumination, decreased salivation, and an increased rate of passage of ingesta, all resulting in decreased abomasal motility and increased gas production.

(3) **Other factors** that are postulated to reduce abomasal tone or motility are hypocalcemia, concurrent diseases through inflammatory mediators (endotoxin and interleukin-1), and lack of exercise. With reduced abomasal motility (atony) or increased gas accumulation, the abomasum distends and may rise dorsally out of place on the left or right side of the animal.

(4) **During late gestation,** it is thought that the gravid uterus pushes the rumen cranially and dorsally, and the abomasum pushes to the left. After parturition, the abomasum moves further left into the void, creating the displacement. This may be more likely in deep-bodied, large cows; thus, a breed or genetic predisposition has been postulated.

(5) **Abomasal displacements in calves** tend to occur at a time when the calf is changing from a monogastric to a ruminant. The pathogenesis is unknown and it is a rare event compared with cattle.

d. Diagnostic plan. It is a relatively straightforward diagnosis in animals that fit the subjective and clinical picture. The diagnosis becomes more suspect in nontraditional animals (calves) or when the ping is transient, recurring, or faint.

e. Laboratory tests

(1) **Clinical pathology tests** are nondiagnostic for the condition but can be helpful, particularly in cases when a ping is not readily auscultable. There may be mild hypocalcemia, hypoglycemia, hypokalemia, ketonemia, ketolactia, and ketonuria.

(2) **Liptak test.** Because other distended viscera produce a ping and may mimic LDA, it is sometimes of value to aspirate fluid percutaneously from just below the most ventral location of the ping. This is known as the Liptak test. Analysis of the fluid retrieved aids in identifying the viscus. If the pH of the fluid is less than 5 and has no evidence of protozoa, it is most likely from the abomasum. If the fluid has a pH of more than 6, it is most likely from the rumen.

f. Differential diagnoses. If LDA is indeed the diagnosis but no ping is auscultable, the condition may be confused with ketosis or simple indigestion. Other conditions that may produce a left-sided abdominal ping and mimic LDA are rumen atony with a gas cap and pneumoperitoneum.

g. Therapeutic plan

(1) **Medical (nonsurgical) management** of the condition is unreliable and usually unsuccessful. However, steps may be taken to treat concurrent disease (e.g., metritis).

(2) **Physical intervention.** Rolling the cow to replace LDA may be undertaken as a temporary treatment in cases where economics do not favor surgery or in cases where the owner wishes to gain some time before salvage. The procedure involves casting the animal into right lateral recumbency, bringing her to dorsal recumbency, rocking her gently back and forth while kneading the ventral abdomen, rolling the cow to her left side, and allowing her to rise. This seems to reposition the abomasum, but a majority of cases recur.

(3) **Surgical intervention** provides for a good to excellent prognosis for a return to economic potential. The following techniques have similar success rates, and choice of technique is a matter of preference or familiarity.

(a) Left flank abomasopexy

(b) Right flank omentopexy

(c) Right paramedian abomasopexy

(d) Right paramedian percutaneous toggle pin (bar suture) fixation

h. Prevention

(1) Management strategies. Many clients tend to live with the problem of LDA as a trade-off because they are satisfied with herd production levels, happy with their feeding management [e.g., total mixed ration (TMR), corn silage], or content with their herd genetics (i.e., large, high-producing cows). The best advice if a client wishes to decrease the incidence of the disease is to incorporate more long-stemmed fiber in the ration, provide exercise for stabled cows, feed animals more frequently with the ration divided, and limit postpartum diseases (e.g., milk fever, metritis).

(2) For the individual cow with LDA, most farmers are able to diagnose the condition themselves after seeing a few cases. However, these clients may need to be reminded that prompt correction of the condition returns the cow to a more economic production unit faster.

2. Right displacement of the abomasum (RDA)

a. Patient profile and history (see I C 1 a)

b. Clinical findings are similar to LDAs, but the ping is on the right side. In the case of a large distention of the abomasum, the displaced viscus may be felt per the rectum.

c. Etiology and pathogenesis (see I C 1 c)

d. Diagnostic plan. The diagnosis is based on clinical findings. An aspirate of fluid from within the viscus (e.g., a Liptak test) may be beneficial for diagnosis.

e. Differential diagnoses. Other conditions that produce a right-sided ping include:

(1) Colonic gas
(2) Gas in the rectum
(3) Cecal dilation
(4) Cecal torsion
(5) Abomasal torsion
(6) Pneumoperitoneum
(7) Physometra

f. Therapeutic plan

(1) Medical management may be attempted with smooth muscle stimulants, but surgery is the treatment of choice. Some sources think that surgety should not be delayed because the RDA might progress to an abomasal torsionlvolvulus. Rolling the cow is contraindicated in RDA cases because an abomasal torsion/volvulus may result.

(2) Surgery is either a right flank omentopexy or a right paramedian abomasopexy.

3. Right abornasal volvulus (right torsion of the abomasum, abomasal torsion)

a. Patient profile and history. Subjective findings are similar m LDA and RDA, although in many cases, an abomasal volvulus may occur in animals other than the early postpartum cow. Sudden agalactia and anorexia are indications.

b. Clinical findings

(1) Appearance. The animal is markedly depressed and may show bruxism or grunting. There may be some evidence of pain or discomfort, such as treading, but this condition is not as painful as other intestinal accidents.

(2) A high-pitched ping is evident over a large area on the right side from the eighth rib to the paralumbar fossa. Fluid may be succussed within the dilated viscus. The cow will be dehydrated as evidenced by a skin tent and sunken eye. Tachycardia is evident (usually 100 bprn], and the pulse is weak.

(3) There is complete rumen stasis possibly with a mild bloat on the left from a gas cap. Feces are scant. There may be only mucus in the rectum, and occasionally, with a concurrent abomasal ulcer, there may be melena.

(4) Rectal examination. The volvulus often can be palpated rectally in the right abdomen. RDA can progress to a volvulus by either a clockwise or counterclockwise twist (when viewing the animal from the side). A torsion can occur around the long axis of the abomasum. Various combinations can be found. If twisted long enough or to a large enough degree, the abomasum becomes congested, hemorrhagic, and infarcted due to occlusion of gastric circulation. As the organ becomes devitalized, the cow goes into shock and dies.

(5) Acid–base changes accompany an abomasal volvulus.

(a) In the early condition, abomasal outflow of HCl is compromised, and reflux occurs into the rumen. The rumen effectively traps H^+ and Cl^- ions. K^+ ions are lost in the urine in deference to H+-ion retention, creating in total a hypochloremic, hypokalemic, metabolic alkalosis.

(b) In the advanced condition, a metabolic acidosis may be found related to the state of shock of the animal.

(6) Occasionally, the omasum is involved in the abomasal volvulus (omasal–abomasal volvulus), which increases the gravity of the prognosis.

c. Etiology and pathogenesis. Dilation is thought to precede torsion of the volvulus and is hypothesized to be caused similarly to LDA and RDA. Mechanical events may cause torsion in the volvulus of the distended abomasum.

d. Diagnostic plan. Diagnosis of the condition is based on clinical findings and laboratory tests. A Liptak test is helpful in determining the affected organ.

e. Laboratory tests

(1) Laboratory findings that support the diagnosis include:

(a) An aspirate of a low pH fluid from the viscus
(b) A toxic leukogram to varying degrees
(c) A metabolic alkalosis (in late stages, a metabolic acidosis)
(d) Evidence of dehydration and prerenal uremia [PCV, total protein (TP), BUN]
(e) A paradoxical aciduria as K^+ ions are excreted in the face of whole-body, H^+-ion deficiency

(2) As the plasma anion gap increases, the prognosis for recovery decreases.

f. Differential diagnoses. It is necessary to rule out other causes of abdominal pings that result in an animal's sick appearance. Conditions to be ruled out include:

(1) Cecal volvulus/torsion
(2) Mesenteric root torsion
(3) Small intestinal obstructions
(4) Acute diffuse peritonitis

g. Therapeutic plan

(1) Immediate surgical intervention coupled with fluid therapy is indicated.

(a) Surgical approaches are a right flank omentopexy or a right paramedian abomasopexy.

(b) Intravenous fluids include isotonic sodium chloride and dextrose for volume expansion (likely 20–80 L), followed by a slow drip of potassium chloride (1 mEq/kg/hour), sodium chloride, and dextrose. Balanced electrolyte solutions are indicated for shock and metabolic acidosis.

(c) Other supportive treatments, depending on the case, include antibiotics, corticosteroids, and NSAIDs.

(2) Salvage instead of surgery may be indicated if dictated by economics and if the animal will pass antemortem and post-slaughter inspection. The systemic involvement of the animal often dictates against this.

h. Prognosis. The prognosis for an animal depends on the degree of abomasal compromise determined at surgery. In some cases that recover, abomasal torsion is associated with the subsequent development of vagal indigestion.

4. Cecal dilation or cecal volvulus

a. Patient profile and history

(1) Cecal dilation and cecal volvulus are conditions that occur most commonly in mature dairy cattle in the first 2 months postpartum. These conditions are more common in the winter months and show no breed predisposition.

(2) The farmer reports that the cow is partially to completely anorexic with a drop in milk production. The herd ration is often a high grain diet rich in carbohydrates such as corn silage or high-moisture corn. It may be fed as a total mixed ration (TMR). The clinical signs usually are reported as moderately

progressive and not sudden in onset unless a dilation has converted into a torsion, in which case signs deteriorate rapidly.

b. Clinical findings

(1) Cecal dilation. With a cecal dilation, the vital signs are usually normal. The animal may tread, indicating mild pain. Feces may be somewhat loose and decreased in amount. The right paralumbar fossa may be slightly distended, and a ping is auscultated in the right flank caudal to the site for RDA. Rumen motility is decreased in rate and strength. Rectal examination confirms the diagnosis, as the apex of the cecum will be felt at the pelvic inlet.

(2) Cecal volvulus. A cecal volvulus offers more dramatic clinical signs than a cecal dilation. The pulse is elevated, and there is complete agalactia, rumen atony, and absence of manure. The cow exhibits abdominal pain by shifting hind-end weight, kicking at the abdomen, and frequent lying down and rising. The right paralumbar fossa may be distended. A ping can be elicited over a larger area than with a simple cecal dilation, and fluid, splashing sounds may be heard. Rectal examination reveals a distended cecal body (the apex has been twisted and directed cranially).

c. Etiology and pathogenesis

(1) The pathogenesis of cecal distention is thought to be similar to abomasal displacements. It is believed that cecal organisms metabolize carbohydrates that have escaped upper gastrointestinal microbial degradation. VFAs, methane gas, and CO_2 are all produced. The VFAs inhibit cecal motility, and the gases accumulate, producing distention. Cecal dilations are thought to precede torsions.

(2) When distended, the cecum may then rotate clockwise or counterclockwise (when viewed from the right). The amount of rotation determines the degree of vascular embarrassment of the organ and the severity of resulting clinical signs.

d. Diagnostic plan. The diagnosis usually is made on the basis of clinical signs and rectal findings.

e. **Laboratory** tests

(1) Laboratory results usually are normal for simple cecal dilation. Animals with cecal volvulus often have a hypochloremic, hypokalemic, metabolic alkalosis. Other laboratory values reflect varying degrees of vascular compromise and shock.

(2) To differentiate cecal volvulus from abomasal volvulus, it may be helpful to perform a Liptak test. The pH is higher (more than 6) with cecal fluid than with abomasal fluid.

f. Differential diagnoses

(1) For cecal dilations, rule out conditions such as abomasal displacement, ketosis, simple indigestion, and colonic gas.

(2) Cecal volvulus may appear similar to abomasal volvulus, traumatic reticuloperitonitis, mesenteric root torsion, and gastrointestinal accidents (e.g., intussusceptions).

g. Therapeutic plan

(1) Simple cecal dilations are treated conservatively.

(a) Diet and exercise. The roughage component of the diet should be increased and forced exercise employed to increase gastrointestinal motility. Calcium salts are used occasionally to treat low-grade hypocalcemia and increase digestive tract motility.

(b) Surgery. Cecal dilation can be a chronic or recurrent condition. Surgery may be used with some success in these cases.

(2) Cecal volvulus requires surgical intervention. The cecum is approached through the right flank. The cecum is decompressed, and ingesta is removed from both it and the adjacent colon. Recovery is usually good with uncomplicated cases. If part or all of the cecum is devitalized, a partial or complete typhlectomy is necessary. Fluid replacement is also recommended to correct the metabolic alkalosis (sodium chloride, potassium chloride, and dextrose).

h. Prognosis. Depends upon amount of devitalization.

D. Bleeding ulcers and erosions. Blood in the feces of ruminants may be frank (visible and either red or black) or occult (unseen). Frank red blood is indicative of lower intestinal origin, whereas dark blood is from higher in the gastrointestinal tract and has been digested by proteolytic enzymes. Occult blood is from minor bleeding of insufficient amount to cause a change in character of the feces.

1. Type I

a. Patient profile and history. Individual animals suffering from a primary disease process may develop abomasal erosions and ulcers. Dairy cows and weaning age calves seem to be most susceptible. The owner usually notices only the primary disease process (a septic process such as acute mastitis, or an abomasal condition such as LDA or RDA). In calves, the history may be one of poor growth and appetite disturbances.

b. Clinical findings

(1) There may be no clinical signs of abomasal erosions or ulcers. The lesions may only be evident at slaughter of a normal animal or necropsy of an animal dying from a primary disease process.

(2) Alternatively, the clinical signs of a primary disease may be accompanied by signs of abomasal erosion or ulceration. These signs include:

(a) Dark, fluid feces with mild, chronic abdominal pain

(b) Periodic bruxism

(c) A capricious appetite

(d) Intermittent fecal occult blood

c. Etiology and pathogenesis. The ulcers in an individual are usually multiple and small. Possible factors contributing to the development of ulcers include:

(1) Hyperacidity. Gastric acid secretion may be stimulated by histamines, high VFA levels, calcium, or stress.

(2) Corticosteroids, which lower mucosal cell resistance

(3) Direct trauma from fiber (straw, low-quality roughage) or foreign bodies (hairballs in calves)

(4) Abomasal distention, resulting in the tearing of mucosa and exacerbation of the lesion by exposure to HCl

(5) Pasture grazing and associations with rainfall, fertilization, and forage growth

(6) Unknown factors, such as endotoxins and mediator substances (e.g., interleukins)

d. Diagnostic plan. Clinical findings and the demonstration of positive fecal occult blood provide the diagnosis. Fecal blood is best discovered by the use of tablets which test for occult blood.

e. Therapeutic plan

(1) In the case of adult ruminants, the primary disease and erosions are self-limiting.

(2) In younger animals, treatment usually is not attempted; however, the owner should pay attention to providing a diet composed of more easily digestible roughage.

(3) Laxatives, such as mineral oil, may be used in the case of concomitant foreign bodies (e.g., hairballs). Alternatively, surgery may be necessary.

f. Prevention. The erosions and ulcers are self-limiting when the primary disorder has been corrected.

2. Type II

a. Patient profile. These ulcers occur in adult cattle usually within 2 months of parturition and are commonly associated with a concurrent abomasal disease (e.g., LDA).

b. Clinical findings. The animal may die suddenly or present with dark, tarry stools (melena). The cow will be anemic, as evidenced by pale mucous membranes, tachycardia, and weakness.

c. Etiology and pathogenesis. The pathogenesis of bleeding ulcers is speculated to be similar to type I ulcers; however, the ulceration involves a major blood vessel, resulting in blood loss. Lymphosarcoma of the abomasal wall is also a primary cause for erosion and invasion of major abomasal blood vessels.

d. Diagnostic plan. Diagnose the condition based on clinical findings and laboratory support. A test on the feces confirms the presence of blood.
e. Laboratory tests. With significant blood loss, the PCV and TSP is decreased. The anemia is characterized as a blood-loss anemia. If there is concurrent abomasal disease, there may be a hypochloremic, hypokalemic, metabolic alkalosis. The leukogram may support a diagnosis of lymphosarcoma in the case of abomasal involvement.
f. Differential diagnoses. Blood in the feces may come from any of several sources. The distinction must be made between dark blood and bright red blood.
(1) A duodenal ulcer presents with fecal blood.
(2) Tumors other than lymphosarcoma may bleed (e.g., squamous cell carcinoma of the stomach).
(3) With vena caval thrombosis in cattle, pulmonary abscesses may develop, which erode pulmonary blood vessels in the lung. Blood is coughed up and swallowed, resulting in fecal blood and a positive test finding.
(4) Salmonellosis, coccidiosis, and rectal lacerations all present with red blood in the feces.
g. Therapeutic plan
(1) Therapy is attempted if lymphosarcoma has been ruled out as a cause of the ulcer. With lymphosarcoma, humane destruction is recommended.
(2) Supportive therapy
(a) For gastric ulcers, therapy includes oral astringents, antacids (magnesium oxide at *225* g four times a day for an adult animal), and protectants (e.g., stat, kaolin, pectin).
(b) **H_2-receptor** antagonists and sulfate disaccharides, although efficacious in monogastrics, rarely are employed in large ruminants because of ruminal dilution and cost.
(c) Propantheline bromide (30 mg four times daily in an adult cow) may be recommended to decrease vagal tone.
(d) A blood transfusion is necessary if the animal's PCV is less than 14% and if the heart rate is more than 100 bpm.
(e) Hematinics may be used (iron, cobalt, B vitamins), and **broad-spectrum** antibiotics are often employed.
(3) Bleeding ulcers are often singular. Surgical extirpation of the ulcer may be considered if bleeding continues and a tumor has been ruled out as the cause. Usually, ulcers are not visible from the serosal surface. Therefore, an abomasotomy is required to locate the ulcer.
h. Prognosis. The prognosis for survival is variable for non-tumor-related abomasal ulcers. Cows respond well to blood transfusions. Lymphosarcoma-associated ulcers carry a grave prognosis.
i. Prevention (see I D 1 f)

E. Anterior abdominal pain. Anterior abdominal pain in the mature ruminant usually manifests itself as a set of common clinical findings. There is an obvious painful response when manual pressure is applied to the cranial portion of the ventral abdominal wall, triggering an audible grunt, noticeable tensing of the neck muscles, or tensing of the ventral abdominal wall. A short groan or grunt may also occur spontaneously when an animal stands or walks downhill.

1. Traumatic **reticuloperitonitis** (TRP, hardware disease, traumatic gastritis, traumatic **reticulitis)**
a. Patient profile and history. In North America, the condition is most common in adult dairy cattle and is sporadic in nature. The owner describes a cow that experiences an abrupt drop in milk production and exhibits partial to complete anorexia.
b. Clinical findings
(1) Acute local peritonitis. Animals in the early stages of TRP are reluctant to move and appear "tucked-up." There may be extension of the neck and an arching of the back. The cow may stand with its elbows abducted and hind feet in the gutter to alleviate the abdominal pain. The triceps muscles often tremble.

(a) Vital signs. Cows may have a shallow, catchy respiratory pattern and a decreased abdominal component to the cycle. The temperature and pulse rate are usually elevated (39.5 °C–40.5 °C and 80–90 bpm, respectively). Usually, the cow is dull and depressed.
(b) On gastrointestinal examination, the clinician finds a decrease in rumen motility or complete atony. The feces of affected animals are scant, firm, dry, and mucus covered.
(c) **Rectal** examination reveals a small, firm-feeling rumen. The animal may grunt with pain as the viscera are pressed forward.
(d) Manipulative tests may be of value in localizing the pain. Cows normally ventroflex when pinched over the withers. Cows affected by TRP will not ventroflex and may grunt with resentment of the procedure. Percussion over the xiphisternum with a closed fist, lifting under the thorax with a board held by a person on either side of the animal or pressure applied by a knee to the ventral thorax may all elicit the same grunt.
(2) Chronic and local peritonitis. The acute clinical signs may abate and be replaced by signs of a more chronic process *24–48* hours after the onset of TRP. Animals appear rough and in poor condition. The vital signs may be normal. Gastrointestinal function is depressed, but signs are usually nonspecific. Demonstrable pain is usually not a hallmark of the local peritonitis with chronic TRP.
c. Etiology and **pathogenesis**
(1) Foreign bodies. Cattle are relatively indiscriminate eaters. Pointed, metallic foreign bodies (e.g., nails, wire) may be consumed on pasture or with the ration. The foreign body is deposited in the reticulum and penetrates the reticular wall, usually in an anterior direction. Ruminal fluid and bacteria follow the track of the foreign body and enter the peritoneal cavity, producing an acute, localized peritonitis.
(2) **Sequelae** to reticular penetration by the foreign body include:
(a) Resolution of the infection with or without medical intervention. The foreign body may "fall back" into the reticulum and the animal "walls-OW' the infection site (acute local peritonitis). The bovine has a tremendous ability to isolate infections by fibrous tissue formation.
(b) Acute diffuse peritonitis. The infection cannot be controlled by the previous inflammatory processes, and a fatal infection ensues.
(c) Traumatic hepatitis or **splenitis.** Movement of a foreign body may occur in a direction other than directly cranial, resulting in the involvement of associated abdominal tissues, most commonly the spleen or liver.
(d) **Pericarditis,** pleuritis, or pneumonia resulting from perforation of the diaphragm
(e) Chronic **peritonitis.** A result of fibrous tissue production may be the formation of reticular adhesions and mechanical interruption of rumen motility (see I A 3).
(f) Fatal hemorrhage due to great vessel perforation (rare)
(g) Diaphragmatic hernia caused by the weakening of the diaphragm (rare)
d. Diagnostic plan
(1) The condition is most often diagnosed through the combination of a thorough physical examination and laboratory tests. Ancillary aids to diagnosis may include the use of radiology, a metal detector, or a compass to determine if a magnet is present in the reticulum.
(2) An abdominocentesis is warranted if the diagnosis is equivocal or requires confirmation. It should be performed *3–4* cm to the right of the midline and *5–7* cm cranial to the foramen of the subcutaneous abdominal vein. Other reported sites for abdominocentesis include a point 10 cm to the right of the umbilicus, on the midline just caudal to the umbilicus, or cranial to the udder under the right flank fold.
(3) An exploratory rumenotomy and/or laparotomy may be both diagnostic and therapeutic.
e. Laboratory tests

(1) Hematologic work-up. The complete blood cell count (CBC) usually reveals a neutrophilia with or without a leukocytosis. A left shift will be present. The serum total protein (TP) is elevated because of an increase in the γ-globulin fraction. Fibrinogen levels are elevated (7–9 g/L). A mild hypocalcemia may be found on clinical chemistry. Note that either very early or chronic cases may have normal clinical pathology findings.

(2) The following findings on abdominal fluid analysis support a diagnosis of acute local peritonitis:
(a) A cell population comprised of 40% neutrophils
(b) Significant proportions of immature, toxic, or degenerative neutrophils
(c) Bacteria contained within neutrophils

(3) The differential cell counts on a CBC and cellular morphology findings on abdominocentesis are most significant in forming a diagnosis because normal ranges of both cell numbers and fibrinogen may overlap with values found in cases of TRP (or other causes of acute local peritonitis).

f. Differential diagnoses. Conditions that need to be ruled out include:
(1) Simple indigestion
(2) Rumen acidosis
(3) Displaced abomasum
(4) Abomasal ulceration
(5) Liver abscess
(6) Pyelonephritis

g. Therapeutic plan
(1) Management strategies. Keep the animal confined in a headgate for 10 days. The front end of the cow may be elevated slightly (20 cm) on a bedding pack. A magnet should be administered orally if there is no evidence or history of one being previously administered.
(2) Antibiotics should be administered according to labeled dosages. Penicillin or broad-spectrum antibiotics may be used. Balanced, isotonic solutions containing added calcium may be administered if the animal is dehydrated.
(3) A **rumenotomy** should be considered to remove the penetrating object(s) if conservative management fails and economics warrant surgical intervention.

h. Prevention of the condition includes administration of magnets to heifers when confirmed pregnant, passing the dairy ration component of the diet over magnets before feeding, and critical attention to pasture or dry-lot management to eliminate sources of foreign bodies (e.g., nails, wire).

2. Perforating abomasal ulcers with localized peritonitis. Abomasal ulcers may perforate the serosa, producing an acute local peritonitis. These may be referred to as type III ulcers (instead of bleeding ulcers).

a. Patient profile and **history.** Abomasal ulcers in mature ruminants most commonly occur in association with concurrent diseases (e.g., LDA), recent parturition, heavy lactation, and grain or pasture grass feeding.

b. Clinical findings. Clinical signs resemble those of TRP (see I E 1 b). However, the pain is localized to the right side of the xiphoid. Careful palpation and ballottement is required to determine this distinction. Bleeding into the gastrointestinal tract rarely seems to accompany a perforated abomasal ulcer.

c. **Etiology** and pathogenesis (see I D 2 c).

d. Laboratory tests. Laboratory data are similar to the acute local peritonitis of TRP (see I E 1 e). There may be an associated hypochloremic metabolic alkalosis if ulcers are concurrent with LDA.

e. Differential diagnoses. Consider other causes of acute local peritonitis on a list of differential diagnoses (e.g., TRP). Also consider conditions that may appear to mimic anterior-abdominal pain, such as:
(1) Liver abscess
(2) Pericarditis
(3) Pleuritis
(4) Endocarditis

(5) Musculoskeletal conditions (e.g., degenerative joint disease, vertebral body diseases, laminitis, tetanus)

f. Therapeutic plan
(1) Management strategies. Provide stall rest, and feed only good quality hay for 10–14 days. If ketosis becomes a problem, add coarse calf grain to the ration.
(2) Antibiotic therapy should be provided as for TRP [see I E 1 g (2)]. Antacid therapy, H_2-receptor blocking agents, and mucosal protectants may be recommended.
(3) If medical therapy is unsuccessful, surgical intervention with removal of adhesions and resection of the ulcer(s) may be employed in valuable animals.

g. Prognosis
(1) The prognosis for abomasal ulceration is usually good; however, it worsens if the lesion is in the abomasal fundus because a pyloric stricture with digestive outflow obstruction may occur. Also, an acute local peritonitis is less likely to be controlled in late pregnancy because of the pressure of a large, gravid uterus. The inability of an animal to control and localize a peritonitis results in an acute diffuse peritonitis with almost invariably fatal consequences.
(2) Occasionally, animals suffer from a chronic, relapsing peritonitis if adhesions break down and release bacteria or their products into the abdominal cavity.

h. Prevention (see I D 1 f)

II. BOVINE INTESTINES

A. Gastrointestinal pain (colic)

1. Adult cattle

a. Patient profile and history. Most colics are caused by either a malposition of a gastrointestinal tract component or other causes of acute obstruction to outflow. Surgery is the treatment of choice.

b. Clinical findings
(1) Appearance and vital **signs.** The animal exhibits a painful abdomen by treading, kicking at the abdomen, stretching, and frequently lying down and rising. The pulse and respiratory rates are elevated. Auscultation of the abdomen reveals absence of both ruminal and intestinal motility. With abomasal or upper intestinal obstruction, the abdomen is slightly distended, and splashing sounds can be elicited by succussion in the lower right quadrant
(2) On simultaneous auscultation and percussion, pings may be evident over distended viscera having a fluid/gas interface. The pings may differ in intensity, location, pitch, and area of auscultation, depending on the source of the distention.
(3) On rectal examination, the rectum is usually empty except for a thick, tenacious, dark red mucus. Insertion of the arm may cause pain or vigorous straining. The examiner may be able to palpate distended or displaced bowel or tight mesenteric bands.
(4) The animal may be dehydrated or in shock. The more proximal the lesion in the gastrointestinal tract, the more rapid the deterioration of the patient

c. Etiology and pathogenesis
(1) The pathogenesis of abomasal torsion/volvulus and cecal torsion/volvulus seems to follow the same course of events as simple dilatations. However, twisting of the dilated viscus on either its horizontal or vertical axis results in digestive outflow *obstruction* and venous vascular occlusion.
(2) The risk **factors** or causes of other intestinal accidents are less clearly understood and less easily studied because of their sporadic nature.
(3) Intestinal accidents
(a) Indications include persistent abdominal pain, elevated heart and respiratory rates, abdominal distention, absence of feces in the rectum, and the presence of a gas-filled viscus or distended loops of intestine.

(b) Intestinal accidents that involve the abomasum or intestinal tract and produce a common subset of clinical findings include:
(i) Right abomasal volvulus
(ii) Small intestinal intussusception
(iii) Mesenteric torsion
(iv) Intestinal incarceration
(v) Cecal dilatation/volvulus
(vi) Stricture of the colon
(vii) Colonic obstruction (e.g., enterolith)
(viii) Colonic constriction (e.g., fat necrosis)

d. **Diagnostic** plan. Perform a thorough physical examination. The presumptive diagnosis is confirmed by surgery.

e. Laboratory tests. Laboratory information is seldom available in time to be of value in the diagnosis.
(1) Hematologic work-up. Shock and dehydration is supported by an increased PCV, increased TP, and a high BUN (prerenal uremia). High duodenal obstruction or abomasal volvulus produces a hypochloremic, hypokalemic, metabolic alkalosis. Leukopenia and/or neutropenia is found with devitalization of infarcted intestine.
(2) Abdominocentesis findings may be variable but often show increased red blood cells, increased leukocytes, and bacteria indicative of devitalized bowel.

f. Therapeutic plan. If the animal is to be successfully treated, a decision to perform surgery should be made early and the patient vigorously supported by fluid therapy.

2. **Calves**

a. Patient profile and history. Most frequently, abdominal pain (colic) in calves is caused by conditions of gastrointestinal origin.

b. Clinical findings are variable depending on the site of involvement, duration of the condition, and degree of vascular compromise of the viscus. Common signs of colic include frequent lying down and rising, kicking of the abdomen, bruxism, and stretching.

c. Etiology and pathogenesis. Acute abdominal pain is of sporadic incidence and often associated with congenital problems of the gastrointestinal tract of young calves. Some conditions may be hereditary.

d. Diagnostic plan. A thorough clinical examination is necessary to determine the most likely source of the pain. Ancillary tests include radiography and contrast studies, gastric intubation, rectal probe (for anal or colonic atresia), and ultrasonography.

e. **Differential diagnoses**
(1) Causes to be ruled out include:
(a) Intestinal atresia
(b) Atresia coli
(c) Atresia ani
(d) Abomasal dilatation
(e) Abomasal torsion
(2) Other causes of abdominal distention must be ruled out including bladder rupture and intestinal fluid pooling with neonatal calf diarrhea.

f. Therapeutic plan. Generally, therapies include surgery and fluid therapy for responsive conditions.

B. Diarrhea

1. Acute diarrhea in adult cattle

a. Bovine virus diarrhea (BVD)
(1) Patient profile and history. The majority of animals affected are young cattle (8 months to 2 years of age) often housed in an intensive management environment (e.g., beef feedlot, dairy freestall housing). There is a variable incidence of clinical disease (usually low). Several animals in the herd may show clinical signs.

(2) Clinical findings. The clinical findings may range from inapparent to acute, severe, enteric disease.
(a) Clinically inapparent disease is widespread. Serologic evaluation may reveal 60%–80% of animals exposed and seropositive.
(b) Mild enteric disease may be seen in a large number of animals and presents as mild transient diarrhea, inappetence, depression, and fever. Cattle recover in a few days.
(c) Acute enteric disease presents as fever, profuse watery diarrhea, dysentery, and tenesmus. Discrete oral erosions are present (or may develop within 7 days of diarrhea), and the animal may salivate excessively. Peracute cases may die without evidence of diarrhea. This has also been described as a hemorrhagic syndrome in cattle.
(d) Clinical signs referable to other body systems include mucopurulent nasal discharge, lacrimation, corneal edema and central corneal opacity; coronitis with interdigital erosions, and reproductive or congenital diseases. Abortions and stillbirths in immunologically negative heifers or cows have been associated with the viral infection, as has the birth of weak calves.
(e) Chronic enteric disease (see II B 2)

(3) Etiology and pathogenesis
(a) BVD is caused by a pestivirus of the ***Togaviridae*** family. Calves acquire passive immunity through colostrum of previously infected dams. This passive immunity wanes in 3–8 months. Following an outbreak of BVD, a herd tends to be free from clinical BVD for several years. With addition of seronegative replacement animals, herd immunity diminishes so that a herd is again susceptible to infection.
(b) The pathogenesis of acute, fatal, enteric disease may involve one of two processes.
(i) Infection by a particularly virulent biotype (BVD Type 2)
(ii) If the fetus is infected by a noncytopathic virus in a BVD-negative dam before 125 days gestation, the calf is immunotolerant to the BVD virus with persistent viral infection and shedding. Subsequent exposure of this animal to a cytopathic virus (likely a mutant of the noncytopathic biotype) results in an acute and fatal infection (mucosal disease).

(4) Diagnostic plan. Clinical findings that are helpful in the definitive diagnosis include diarrhea, shallow oral erosions, central corneal opacity, and interdigital erosions.

(5) Laboratory tests. In individual animals, evidence of leukopenia, seroconversion, virus isolation, and polymerase chain reaction (PCR) are helpful for diagnosis. Note that animals that are immunotolerant and suffering from mucosal disease may not seroconvert. Herd serology may aid in diagnosis, as will necropsy of affected animals.

(6) **Differential** diagnoses. Conditions m be ruled out include bovine malignant catarrh, rinderpest, and salmonellosis.

(7) Therapeutic plan
(a) Mild cases. Therapy is not necessary in cases of mild disease and of little help with severe enteric disease. Treatment is of no value in mucosal disease.
(b) Severe cases. When therapy is attempted in severe cases, treatment consists of fluid therapy, oral astringents, and systemic antibiotics to control secondary infections.

(8) Prevention
(a) Isolation of clinically ill animals from naive animals may limit the spread in the case of a herd outbreak.
(b) Vaccination may be practiced with either killed or modified live viruses. Modified live virus vaccines have been limited to nonpregnant animals in their use because of possible induced abortions. It is unsure if this is related to the vaccine virus, vaccine virus reversion, or vaccine

contamination. Beef calves should be vaccinated between 6 and 8 months of age and dairy cattle vaccinated annually.

b. Bovine malignant catarrh (BMC, malignant catarrhal fever)
   (1) Patient profile and history. The condition is usually sporadic and found in single animals, although occasionally herd outbreaks occur. In North America, the history invariably includes contact with sheep (usually at lambing time). In Africa, history includes contact with wildebeest.
   (2) Clinical findings
      (a) High fever with tachycardia accompanies profuse diarrhea and dysentery. Other signs include anorexia, agalactia, mucopurulent nasal discharge, dyspnea, and lymphadenopathy.
      (b) Ocular changes include a peripheral corneoscleral opacity that spreads centripetally, ocular discharges, eyelid edema, and blepharospasm.
      (c) Neurological signs may be seen including incoordination, nystagmus, muscle tremors, head pressing, paralysis, and convulsions.
      (d) Chronic disease produces eczematous lesions around the perineum, prepuce, axillae, and horns. Sloughing of the skin from the teats and vulva is found in acute cases.
      (e) Labeling. This variety of clinical pictures has resulted in labeling the disease by its presenting form (i.e., alimentary form, head and eye form, neurological form, skin form).
   (3) Etiology and pathogenesis
      (a) BMC is a generalized infection of the primitive mesenchyme. Necrosis and proliferation of vascular adventitia (vasculitis) yields epithelial erosions, keratoconjunctivitis, and encephalitis. Lymph node enlargement is caused by atypical proliferation of T lymphocytes.
      (b) Clinical disease occurs only in cattle, deer, and buffalo. Sheep and goats in North America and wildebeest in Africa transmit the virus, although neither of these hosts develop clinical signs. Alcelaphine BMC virus (BHV-3) is the wildebeest-associatedagent. A yet unidentified herpesvirus (sheep associated herpesvirus) is the North American agent. The sporadic nature of the condition makes studying the disease or determining etiologies difficult.
   (4) Diagnostic plan. Diagnosis of the condition is aided by the history (association with hosts), sporadic nature of the condition, and uniqueness of the clinical findings.
   (5) Laboratory tests
      (a) Hematologic work-up. Animals exhibit an early leukopenia followed by a leukocytosis. There are inflammatory changes of the joint fluid and cerebrospinal fluid.
      (b) Serological conversion occurs with the African disease but has not been demonstrated with the North American disease. Virus isolation remains difficult.
      (c) Postmortem examination remains the definitive diagnostic tool.
   (6) Therapeutic plan. Therapy is unrewarding and animals will die in spite of supportive care.

c. **Rinderpest**
   (1) Patient profile and history. This disease is confined to the Middle East, Asia, and tropical Africa where it is enzootic. It affects all ruminants and emerges in outbreaks.
   (2) Clinical findings
      (a) Acute cases. A high fever precedes by a few days other clinical signs such as anorexia, agalactia, and lacrimation. Oral and nasal inflammation soon is followed by the development of coalescing necrotic ulcers. Other signs follow including hemorrhagic and purulent lacrimation, skin lesions, diarrhea, dysentery, and dehydration. In an immunologically naive population, many cattle die acutely, most succumb within 7–10 days and few survive.

      (b) Subacute and chronic forms of the disease occur in more resistant populations and survival rates are higher.
   (3) Etiology and pathogenesis. Rinderpest virus is a paramyxovirus spread by animal excretions. Close animal contact is necessary because the virus does not survive for long periods outside of the host. The virus has a high affinity for lymphoid tissue and alimentary mucosa, where it replicates and produces the focal necrotic stomatitis and enteritis. A strong antibody response is generated.
   (4) Diagnostic plan. Diagnosis is based on clinical findings, history, and postmortem lesions.
   (5) Laboratory tests
      (a) Leukopenia and lymphopenia occur in affected animals. Postmortem findings are highly suggestive in populations at risk.
      (b) Viral antigen may be detected in several excretions and will confirm the diagnosis. Serology is of less value because of the acuteness of the condition in many cases.
   (6) Differential diagnoses. Other diseases that cause oral lesions and diarrhea must be ruled out. It is equally important to differentiate this disease from others that produce similar oral lesions without diarrhea (e.g., foot and mouth disease).
   (7) Therapeutic plan. There in no known treatment for this virus.
   (8) Prevention. Vaccination programs are successful, and many have lead to the eradication of the disease. Total eradication requires regulatory measures ensuring vaccination of herds and limitations on movements of nomadic populations.

d. **Salmonellosis**
   (1) Patient profile and history. This disease occurs worldwide and is seen in all ages, species, and breeds of animals. There is often a history of stress (e.g., recent shipment, parturition).
   (2) Clinical findings
      (a) Vital signs and diarrhea. There is an initial fever followed by a subnormal temperature. The heart and respiratory rates are elevated. There is a severe, watery, hemorrhagic diarrhea with mucus, fibrin casts, and blood clots. There is frequently evidence of abdominal pain (groaning, kicking at the flank), and pregnant animals may abort.
      (b) The disease has been described as one of three syndromes: septicemia, acute enteritis, and chronic enteritis. The main presenting syndrome in cattle is the acute enteritis.
   (3) Etiology and pathogenesis
      (a) The most common species isolated worldwide is Salmonella *typhimurium*, although a variety of Salmonella species can cause disease. S *dublin* has a more patchy distribution but is the most common isolate in Europe.
      (b) The most common source of infection is environmental and feed contamination. Any domestic or wild species of animal or bird can act as a source of infection.
      (c) S *typhimurium* causes sporadic, occasionally fatal disease. Infected adults are carriers for short periods of time so that the disease incidence usually subsides when the source of infection is removed.
      (d) S *dublin* is particularly well adapted to cattle, which may act as a reservoir for outbreaks. Continued excretion of the organism may occur for years after exposure.
      (e) Route of infection. After oral inoculation with the bacteria, salmonella invades the intestinal wall and progresses to localize in the mesenteric lymph nodes. Development of disease then depends on the immune status and age of host, virulence of the organism, and stresses on the animal. In susceptible animals exposed to a virulent species, septicemia and bacteremia occur. A carrier state may develop in survivors. Diarrhea occurs due to enteritis and the elaboration of an enterotoxin, which causes an increased secretion of sodium, chloride, and water into the gut lumen.
   (4) Diagnostic plan. Clinical findings together with laboratory test results usually

are sufficient for diagnosis; however, fecal culture results may be negative even with diarrhea.

(5) Laboratory tests
(a) Clinical pathologic findings reflect a profound toxic state and an inflammatory condition. Anemia from blood loss may be seen.
(b) Fecal culture is the best diagnostic test but may need to be repeated several times for success. Organisms may not be present in the feces for up to 2 weeks after commencement of diarrhea because of the dilution effect of the diarrhea. Culturing a rectal mucosal biopsy increases the likelihood of successfully isolating the organism in an affected animal.

(6) Differential diagnoses. Other conditions that appear similar to enteric salmonellosis include winter dysentery, coccidiosis, general toxemia, and arsenic or superphosphate fertilizer toxicities.

(7) Therapeutic plan
(a) Early, aggressive treatment with broad-spectrum or selective antibiotics is necessary for successful therapy. Extralabel use of these antibiotics requires restrictions on meat and milk use from treated animals. Examples include:
(i) Trimethoprim–sulfa: 25 mg/kg intramuscularly twice per day
(ii) Centamicin: 2 mg/kg intramuscularly three times per day
(iii) Amikacin: 7 mg/kg intramuscularly three times per day
(b) Oral or intravenous fluids are necessary in amounts calculated for replacement and ongoing losses. Oral astringents and protectants (e.g., bismuth subsalicylate) and parenteral nonsteroidal anti-inflammatory agents (e.g., flunixin meglumine) may be employed.

(8) Prevention
(a) Recovered animals (whether from treatment or naturally) may excrete the organism and expose herdmates for significant lengths of time. Treatment and isolation procedures must take this into account.
(b) Management strategies. Seek to remove sources of contaminated food, litter, and water. Salmonella organisms may infect humans, so hygienic precautions should be taken.

e. Winter dysentery
(1) Patient profile and history. This disease commonly occurs in young adult, housed dairy cattle in the winter months. Diarrhea is most severe in lactating and pregnant animals and is rare in bulls and steers. Diarrhea is often related to a feed change, a change in housing, or sudden, significant temperature shifts.

(2) Clinical findings
(a) Herd. This disorder causes an outbreak of diarrhea involving the majority of animals in the herd. There is an accompanying drop in milk production. The condition may persist in the herd for up m 2 weeks.
(b) In individual animals, the TPR is usually normal. There is a nasolacrimal discharge and a cough, both of which precede the diarrhea. The diarrhea is projectile and hemorrhagic and lasts from a few hours to a week. The animal may demonstrate abdominal pain, have increased intestinal sounds, and become dehydrated and weak.

(3) Etiology. The cause is considered to be a bovine coronavirus.

(4) Diagnostic plan. The clinical picture and subjective findings along with response to therapy are enough to establish a diagnosis.,

(5) Laboratory tests. Individual animals have a mildly elevated hematocrit and total plasma protein value. Direct electron microscopy and an enzyme-linked immunosorbent assay (ELISA) may be applied to the feces to demonstrate the virus. Acute and convalescent serum samples reveal seroconversion to coronavirus.

(6) Therapeutic plan. If necessary, animals may be treated with oral or intravenous fluids and oral intestinal protectants (e.g., kaolin).

(7) Prevention. Even with care regarding disinfection and attempts at isolation, the disease will run its course throughthe herd.

f. Arsenic toxicosis
(1) Patient profile and history. Recent exposure to arsenicals (e.g., ectoparasite sprays, arsenic-based herbicides, arsenic-based wood preservatives) may be deduced from the history. The owner may report sudden death of an individual or group of animals.

(2) Clinical finding
(a) Animals may experience acute, subacute, or chronic signs referable to arsenic poisoning. With the acute toxicosis, cattle experience abdominal pain, diarrhea, dehydration, regurgitation, muscular tremors, convulsions, and death within 4–6 hours of showing clinical signs.
(b) Other signs may involve the central nervous system (CNS; see Chapter 11).

(3) Etiology and pathogenesis. Ingested inorganic arsenic causes inactivation of sulfhydryl groups in tissue enzymes. Tissues that are most susceptible are the alimentary tract, liver, kidney, spleen, and lung. In the gastrointestinal tract, this condition causes extensive capillary damage, hemorrhage, necrosis, and sloughing of the intestinal mucosa.

(4) Diagnostic plan. Clinical findings and history are important, but diagnosis relies on laboratory confirmation.

(5) Laboratory tests. Urine and hair samples are suitable antemortem specimens for arsenic analysis. Postmortem confirmation is best supported by liver arsenic levels.

(6) Therapeutic plan
(a) An attempt should be made to absorb the enteric arsenic with activated charcoal at 1–4 g/kg orally. Cattle are also treated with sodium thiosulfate at 15–30 g in 200 ml H_2O intravenously followed by 30–60 g orally, four times daily. Treatment should be continued until recovery occurs.
(b) British antilewisite (BAL), also called dimercaprol, may be used but is less effective against inorganic salts than the organic forms. Intravenous fluid therapy is warranted in dehydrated animals.

(7) Prevention. Limit exposure to arsenicals.

g. Toxemia. Toxemia, such as peracute coliform mastitis or toxic metritis in cattle, often are accompanied by diarrhea. The pathogenesis may be an effect of endotoxemia or caused by stress-relatedulcers.

2. Subacute to chronic diarrhea in young and adult cattle
a. Chronic parasitism. This condition is common, but diarrhea due to parasitism is relatively infrequent Chronic wasting is the obvious sign, and of the parasites affecting the bovine abomasum and small intestine (e.g., Ostertagia, *Cooperia, Trichostrongylus, Nematodirus*), *Ostertagia* infestation produces the diarrhea.
(1) Patient profile and history. Several young (6 months to 2 years) animals often are affected in a herd. There is a history of persistent diarrhea and weight loss.
(2) Clinical findings. There is diarrhea without odor, mucus, or blood. Emaciation, dependant edema, and poor growth also may be signs of infection.
(3) Etiology and pathogenesis
(a) Type I. Infective (third-stage) larvae are ingested, molt to fourth-stage larvae, and penetrate the abomasal glands, causing hyperplasia of the mucus-secreting cells. This nodular hyperplasia may be discrete or confluent, presenting as a "moroccan-leather" appearance on necropsy. Mucosal layer destruction results in protein leakage.
(b) Type II. Fourth-stage larvae enter the mucosal glands and remain there, causing little or no damage (a pretype condition). Type II disease occurs when larvae emerge, producing marked cellular hyperplasia. This emergence occurs at various times of the year in different countries. Abomasal pH rises due to a loss of parietal cells and failure of the conversion of pepsinogen to pepsin. Anorexia and plasma protein leakage cause hypoproteinemia. Diarrhea is constant and weight loss is rapid.
(4) Diagnostic plan. Clinical findings, environmental findings, and investigation of management practices may indicate a parasite burden.

(5) Laboratory tests. Fecal egg counts are diagnostic, however, they may need to be repeated in the case of hypobiotic larvae.
(6) Therapeutic plan. Deworm in the event of any parasite load. Ivermectin and levamisole continue to be the drugs of choice (ivermectin for the hypobiotic state).
(7) Prevention. Reduce pasture or drylot contamination through management of stocking densities, pasture rotation, and routine anthelmintic treatment.

b. Chronic BVD
(1) Patient profile and history. The age range of affected cattle is usually 6 months to 2 years. There may be a history of a previous outbreak of BVD in the herd with the recovery of most animals. A few animals may have remained stunted with intermittent diarrhea, lameness, or both. There may be a history of recurrent bacterial infections (e.g., pneumonia).
(2) Clinical findings. Animals appear stunted with a rough, dry haircoat. Oral examination reveals the occasional erosion with blunted oral papillae. Crusty or erosive dermatitis is present at the commissures of the mouth, medial canthus of the eye, around the perineum, scrotum, coronary band, bulbs of the heels, and in the interdigital cleft. The animal may be lame. Diarrhea is intermittent or continuous.
(3) Etiology and pathogenesis. Cattle previously exposed to a noncytopathic strain of BVD virus in utero (before 125 days gestation) are immunotolerant to the BVD virus and are incapable of mounting a humoral antibody response when subsequently challenged with the virus. They are chronically infected with the virus, continue to excrete antigen, but remain negative by serum neutralization tests.
(4) Diagnostic plan. Diagnosis may be based on clinical findings, virus isolation or PCR for viral nucleic acid, and necropsy findings.
(5) Therapeutic plan. Therapy is useless and salvage should be considered as a realistic option.
(6) Prevention. Recommendations for control are as presented in II B 1 a (8).

c. Coccidiosis
(1) Patient profile and history. Subjective findings with this disease are similar to conditions predisposing to parasitism (i.e., overcrowded housing conditions, management practices that encourage fecal–oral spread of organisms). Groups of young animals are affected in a seasonal pattern depending on region of the country.
(2) Clinical findings. The cardinal signs are diarrhea (with mucus and blood) and tenesmus. Anemia also may be a finding, and some cattle may have nervous signs (see Chapter 11). Cattle often are unthrifty in appearance. Clinical syndromes may vary from peracute cases to inapparent infections.
(3) Etiology and pathogenesis
(a) Causative organisms are ***Eimeria zuernii*** and E. ***bovis*** in the large intestine and ***E. ellipsoidalis*** in the small intestine.
(b) Infestation occurs through the ingestion of sporulated oocysts from the environment. Oocysts are resistant to most environmental conditions. Multiple-species infections are quite common, and disease seems to be more prevalent in stressed or undernourished animals.
(c) Route of infection. Sporozoites are released from the ingested oocytes and invade the endothelial cells of the small intestine. Asexual schizonts develop, mature, and release merozoites through rupture of the endothelial cells. This cycle repeats itself in the large intestine followed by the sexual life cycle of macrogametocyte and microgametocyte production. These stages of the life cycle also produce intestinal mucosa destruction. Fertilization of the gametocytes produce oocysts, which are shed coincident with the development of diarrhea and dysentery. The prepatent period (and development of diarrhea) may be 15–20 days.
(4) Laboratory tests. Oocysts are seen in the feces 2–4 days after the onset of dysentery. Therefore, clinical signs may be present without demonstrable oocysts. *Coccidia* may be found in the feces of normal calves, so laboratory data must correlate to clinical findings. Direct fecal smears and fecal flotations are the most common laboratory tests used.
(5) Therapeutic plan
(a) Individual treatment. Coccidiosis is a self-limiting disease but causes death in severely affected animals. Clinical signs subside when the multiplication stages have passed. Most coccidiostats suppress early first-stage schizonts; therefore, treatment of clinical disease with coccidiostats is of limited value. Other supportive care for the individual animal (e.g., fluid therapy) may be warranted under certain circumstances.
(b) Herd treatment. Mass medication, decreasing stocking densities, and removing feed and water from ground level are all treatment strategies used in a herd outbreak. Medications include sulfaquinoxaline at 2.72 mg/kg orally daily for 3–5 days and amprolium at 10 mg/kg orally for 5 days.
(6) Prevention. Avoid undernutrition, overcrowding, and feeding from ground level. Coccidiostats may be efficacious and necessary under conditions that do not allow for management alterations. Control medications include sulfaquinoxaline, amprolium, decoquinate, monensin, and lasalocid.

d. Chronic salmonellosis. Salmonellosis in adult cattle usually presents as an acute, explosive, mucohemorrhagic diarrhea (see II B 1 d). However, when salmonellosis has become enzootic, chronic diarrhea and unthriftiness may be observed.

e. **Johne's** disease **(paratuberculosis)**
(1) Patient profile and history. This disease occurs in adult dairy cows and beef cattle older than 2–3 years of age. There is a breed disposition, with Shorthorn, Angus, and Channel Island dairy breeds being over-represented. This may be an effect of historical numbers possibly related to breed popularity, similar to the way Holsteins are affected now.
(2) Clinical findings. Early clinical disease presents as intermittent or continuous watery or "pea-soup" diarrhea associated with stress (e.g., parturition). Vital signs are normal, and appetite is good to excellent. Emaciation becomes progressive, and milk production decreases. Terminal signs include profound emaciation, profuse watery diarrhea, and dependent edema.
(3) Etiology and pathogenesis
(a) The disease is caused by ***Mycobacterium paratuberculosis.*** The infection is contracted principally by the ingestion of feedstuffs or water contaminated by animals that are fecal shedders. Nursing calves may ingest the organism off the dam's udder. Calves born to clinically affected cattle may be infected in utero.
(b) Age of infection. Animals are most susceptible to infection as calves (less than 30 days of age), and most animals with the disease are infected before 4 months of age. However, clinical disease develops 2–5 years later.
(c) Age of shedding. Animals may become fecal shedders 15–18 months before the development of clinical signs. Some animals are shedders without ever becoming clinically affected. The organism may be persistent in suitable soils.
(d) Route of infection. Following oral ingestion, M. *paratuberculosis* localizes in the small intestine and associated lymph nodes. The bacteria multiply, and the animal either becomes resistant, a shedder, or a clinical case. Animals that are infected older than 6 months of age apparently are able to mount an effective immune response and develop resistance.
(e) Disease progression. In those animals in which the organisms multiply unchecked, there develops a granulomatous enterocolitis initially around the ileocecal valve. The enterocolitis spreads, and a malabsorption syndrome results with a protein-losing enteropathy. There is a compensatory increase in protein synthesis by the liver, but eventually hypoproteinemia occurs with signs of edema.
(4) Diagnostic plan. Clinical signs alone are not reliable for a diagnosis. Laboratory tests confirm the diagnosis. Postmortem examination of a specifically sacrificed animal is the most reliable diagnostic tool.
(5) Laboratory tests

(a) Fecal culture is the most reliable and frequently used diagnostic test for infection in individual cattle. DNA probes and fecal culture by radiometric technique are also available and considered valuable diagnostic tools.
(b) Serological tests offer some value in determining herd status of infection but often must be carried out as a series of tests. Problems with sensitivity and specificity are common. These tests include complement fixation test (CFT), agar gel immunodiffusion test (ACID), and ELISA (the least costly and most accurate serological test).
(c) Other tests. Tests for cell-mediated immunity vary in their reliability. The intradermal and intravenous johnin tests are available. Lymphocyte immunostimulation tests are also available, of which some are highly reliable.

(6) Therapeutic plan. Therapy is as yet unwarranted because response to treatment with medications (e.g., streptomycin, isoniazid, clofazimine) is only transient.
(7) Prevention
(a) Methods of herd management. The disease may be kept in check on a herd basis by test and slaughter methods. Removal of reactor or culture-positive animals and their offspring is necessary. This needs to be combined with improved hygienic practices, which decrease the fecal–oral spread of the organism. Calves must be removed from the dams at birth and reared separately. The herd should be maintained as a closed herd or replacement animals purchased from known Johne's-free herds.
(b) Problems with herd management. The disease may be effectively eliminated from a herd, but the management changes required are intensive, rigorous, and expensive. Because of the inaccuracies in diagnostic tests, there is as yet no test and slaughter program that effectively eliminates all carriers. Therefore, the only effective method of elimination is one of repopulation of a new environment with unexposed or known negative animals.
(c) Vaccination. If local legislation permits, vaccination of calves may provide protection against clinical disease and reduce the rate of spread of infection. A major complication of a vaccination program is that vaccines are positive to the tuberculin test for bovine tuberculosis.

f. Bovine leukosis (enzootic bovine leukosis). Bovine leukosis is of major significance, but the gastrointestinal effects are of limited importance to the overall clinical picture and health management of the disease.
(1) Patient profile and history. The disease is seen most commonly in adult dairy cows. History related to gastrointestinal signs includes intermittent diarrhea.
(2) Clinical findings. The clinical signs depend on the degree and specificity of organ involvement. Many of the following forms often occur concurrently, resulting in the term adult multicentric lymphosarcoma.
(a) Digestive form—capricious appetite, persistent or intermittent diarrhea, melena
(b) Lymph node form—superficial, palpably enlarged lymph nodes
(c) Cardiac form—muffled heart sounds due to hydropericardium, dyspnea due to hydrothorax, jugular vein engorgement, brisket edema, and bottle jaw
(d) Nervous form—posterior paralysis
(e) Respiratory form—upper respiratory noise and dyspnea
(f) Ocular form—exophthalmos
(3) Etiology and pathogenesis
(a) The pathogenesis of the various forms is covered under the appropriate systems. Gastrointestinal signs are a result of tumor growth in the abomasal wall. Ulceration and gastrointestinal bleeding follows.
(b) Etiology. Enzootic bovine leukosis is caused by the bovine leukemia virus (BLV), a type-C retrovirus. Infection with the virus is common, but the development of solid tissue tumors is less common, depending on host genetic and environmental factors. Persistent lymphocytosis and bovine lymphosarcoma are manifestations of the endemic form of bovine leukosis. The usual incubation period between infection and development of clinical signs is 4–5 years.
(c) Horizontal transmission via blood or contaminated instruments, which transmit infected lymphocytes, is thought to be the most common method of infection. Therefore, insect bites, surgical instruments, rectal sleeves, and contaminated needles have all been implicated in viral transmission.
(d) Vertical transmission is possible through contaminated semen as is transplacental exposure to the virus during gestation. There is a familial tendency in the development of disease, indicating the possibility of a genetic predisposition.

(4) Diagnostic plan. The diagnosis of bovine lymphosarcoma as causative for gastrointestinal disease relies on definitive tests, necropsy or exploratory laparotomy with abomasotomy. Clinical findings of diarrhea with palpably enlarged lymph nodes or other multicentric expressions increase the level of suspicion.
(5) Laboratory tests. Laboratory tests that support a diagnosis include abdominocentesis demonstrating exfoliated, abnormal lymphocytes; serology (ACID, radioimmunoassay, ELISA, protein immunoblot test); persistent lymphocytosis; and solid tumor biopsy.
(6) Therapeutic plan. There is no known treatment for this disease.
(7) Prevention. Disease may be controlled or eradicated.
(a) Control is based on a test and segregation practice. Animals are tested and divided into positive and negative groups, and they are distinctly separated in terms of housing and management. In one such scheme, AGID tests are used on a herd basis every 3 months and reactors removed to the positive group until reactors are no longer found. Testing is then used every 6 months until all animals have been negative for four successive tests. Replacement animals must be test negative on two separate occasions and quarantined for 60 days before the second test.
(b) Eradication is effective but costly. Infected animals are identified by ACID. Seropositive animals are culled. This process is repeated every 30–60 days until the herd tests clean. Testing is then carried out every 6 months until four sequential negative herd tests allow the herd to be declared BLV free.
(c) Several procedures help limit the spread of infection within herds including
(i) Feeding colostrum to newborn calves from BLV-free animals
(ii) Housing calves in individual hutches
(iii) Control of insect vectors
(iv) Dedication of blood-bearing fomites to individual use (e.g., rectal sleeves, needles)
(v) Disinfection of veterinary instruments
(vi) Embryo transfer

g. Abdominal fat necrosis
(1) Patient profile and history. Although uncommon, this condition is most prevalent in obese Channel Island cattle (Jersey, Guernsey). Often sporadic in occurrence, the disease has been associated on a herd basis with cattle grazing heavily fertilized, fescue pastures.
(2) Clinical findings
(a) Clinical signs include diarrhea or findings reflective of intestinal obstruction (i.e., anorexia, treading, stretching, bruxism, lowered fecal output, abdominal distention).
(b) Rectal examination reveals large, irregularly-shaped masses associated with the kidneys, small intestine, and colon. Rectal stricture may be evident. Dystocia may occur secondary to the abdominal masses.
(3) Etiology and pathogenesis. Fat deposits in the body are modified by a coagulative necrosis. This process is associated with over-conditioning in individuals or linked to high nitrogen levels in fescue pasture on a herd basis. The fat deposits harden and interfere with gastrointestinal function.
(4) Diagnostic plan. Diagnosis is based on rectal examination and necropsy.

(5) Therapeutic plan. There is no approved treatment for this disorder, although the condition may resolve if the animals are taken off an offending pasture. Isoprothiolane has shown experimental efficacy. Salvage is recommended.

h. **Primary** copper deficiency

(1) Patient profile and history. The condition usually occurs in young adult cattle (1–3 years) that graze on sandy or peat soils. The condition presents as a herd problem. There is no breed predisposition.

(2) Clinical findings. In clinically evident cases, animals are consistently poor in appearance with persistent diarrhea. Occasional depigmentation is evident around the eyes. Lameness may be evident as is anemia, decreased milk production, or occasionally sudden death. CNS signs (e.g., incoordination, paresis) occur in lambs (see Chapter 11).

(3) Etiology and pathogenesis. Copper may be inadequate in the diet because of soil deficiency or unavailability. Lack of copper limits the cytochrome oxidase system through decreased production of ceruloplasmin. Failure of these enzyme systems result in the many manifestations of copper deficiency.

(4) **Diagnostic** plan. The diagnosis may be supported by laboratory data (generated from h'erd-level sampling) and response to treatment.

(5) **Laboratory** tests. Plasma and tissue (liver, hair) copper levels are low, but interpretation may be difficult because of wide variations in values for individual animals. Anemia is evident on a CBC. The diet may be analyzed for copper levels.

(6) Therapeutic plan

(a) Oral dosing of 4 g of copper sulfate for young animals and 8–10 g of copper sulfate for mature cattle weekly for 3–5 weeks is recommended. Controlled-release boluses are also available, but absorbed amounts may be less than desirable. Copper oxide fragments are available for oral dosing and may be the most reliable and efficacious method of supplementation.

(b) Injectable copper treatments may provide advantages over oral forms in terms of ease in administration and rapidity of results.

(7) Prevention. Treatment must be followed by oral maintenance. Oral dosing weekly with 5 g/adult or dietary supplementation in the mineral mix is recommended. Salt licks containing 2% copper sulfate should be adequate. Dietary modification to ensure 10 parts per million (ppm) copper as measured in the dry matter of the final ration is also adequate. Top dressing of the pasture with copper sulfate at a rate of 10 kg/ha may be employed.

i. Secondary copper deficiency (molybdenum toxicity)

(1) Patient profile and history. This deficiency affects young adult dairy cows and beef cattle grazing pastures high in molybdenum or sulfates.

(2) Clinical findings. The clinical picture is as in primary copper deficiency, but anemia is not as common. Diarrhea (watery, yellow-green to black feces passed without straining) is more common than in primary copper deficiency.

(3) Etiology and pathogenesis. Molybdenum reacts with sulfides in the rumen to produce thiomolybdates. The subsequent formation of copper–thiomolybdate complexes renders the copper unavailable. Soil levels of less than 3 ppm molybdenum are considered safe, whereas offending soils are frequently in the 10–100 ppm range.

(4) Diagnostic and therapeutic plans. Diagnosis and treatment are as for primary copper deficiency (see II B 2 h). The removal of sulfates from drinking-water may have a positive effect.

(5) Differential diagnoses. Rule out parasitism.

(6) Prevention centers around removing copper-complexing agents (e.g., sulfates) from the diet and water source.

3. Diarrhea in neonates

a Enterotoxigenic ***Escherichia coli*** (ETEC)

(1) Patient profile and history. Diarrhea is a common occurrence in young calves of all breeds. Diarrhea due to *E. coli* occurs often at less than 5 days of age but can be seen in older neonates (up to 2 weeks).

(2) Clinical findings. Diarrhea is watery and profuse, leading to depression, weakness, dehydration, and anorexia. Terminally, there are signs of shock resulting from hypovolemia and electrolyte imbalances. Mild cases may recover spontaneously in a few days, whereas severe cases may result in death in as little as 8 hours (sometimes without external evidence of diarrhea).

(3) Etiology and pathogenesis

(a) Pathogenesis. ETEC adhere to the intestinal epithelium via pili, which usually possess the K-99 antigen. When adherent, ETEC produces a heat stable toxin (ST). This results in a secretory diarrhea (loss of fluid and electrolytes), mediated by cyclic guanosine monophosphate (cGMP). The ST leaves the glucose (glycine)-Na^+ transport system intact but interferes with the Ca^{++}-mediated Na^+ Cl^- co-transport system.

(b) In addition to the bacteria, contributing factors to the development of diarrhea include:

(i) Intensive management conditions (e.g., overcrowding, communal feeding)

(ii) Synergism with other diarrhea-producing agents (e.g., rotavirus)

(iii) Ingestion of insufficient quantities or substandard qualify of colostrum

(4) Diagnostic plan. Clinical findings must be supported by a laboratory diagnosis to allow appropriate recommendations to be made regarding herd prevention.

(5) Laboratory tests

(a) Fecal or **intestinal** culture reveal ETEC often based on polyclonal antibody testing. ELISA test kits for demonstration of the K99 antigen are also available.

(b) For the individual calf, laboratory findings show varying degrees of acidemia, hypoglycemia, and hypokalernia. Clinical pathology findings are reflective of hemoconcentration (e.g., elevated BUN and hematocrit). Total protein levels may be normal if hypoproteinemia is coincident with dehydration.

(6) Therapeutic plan

(a) Antibiotics. Diarrhea caused by ETEC is often self-limiting without antibiotic therapy if vigorous supportive care is instituted early. However, antibiotics often are used with success in the field (e.g., sulbactam–ampicillin, trimethoprim–sulfas, gentamicin). Part of this rationale is because of the difficulty of accurately differentiating between septicernic colibacillosis and diarrhea due to ETEC.

(b) Supportive care for the individual calf is an absolute necessity. Oral replacement solutions are efficacious early in the course of the disease. Later, it becomes necessary to replace lost fluids and electrolytes intravenously.

(i) Oral replacement solutions work on the principle of an intact glucose absorption mechanism in the gut. Sodium is absorbed via coupling to glucose, and water is dragged along the osmotic gradient

(ii) Replacement solutions contain sodium chloride, sodium bicarbonate, and an energy source (e.g., glucose). The oral agents should be nursed by the calf in small quantities frequently, based on replacement needs and ongoing losses. It is not necessary to take calves completely off milk, but decrease quantities and alternate with electrolyte feedings. Do not feed oral electrolytes and milk simultaneously as this may interfere with milk clot formation in the a b omasum.

(iii) In calves more than 8% dehydrated, intravenous replacement and alkalinizing solutions are necessary. Isotonic $NaHCO_3$ (1.3%) is often administered in conjunction with balanced electrolyte solutions (0.85% sodium chloride) and isotonic dextrose (5%).

(c) Other therapies include nursing care to keep the calf warm and dry and intestinal protectants (e.g., kaolin–pectin combinations, bismuth

subsalicylate). There is no evidence that anticholinergics or oral antibiotics influence the course or magnitude of the diarrhea.

(7) Prevention. Total prevention of this condition is usually an unrealistic goal. A control program is built on the principles of reduction of exposure of neonates through hygienic and management practices, provision of adequate colostrum, and vaccination of the dam or calf. Many of these practices are difficult to fully achieve and require creative modifications, depending on the numbers of animals and the population at risk (i.e., dairy, beef, or veal calves).

b. **Rotavirus** diarrhea

(1) Patient profile. Rotavirus causes diarrhea in all breeds of calves from 5 to 7 days up to 3 weeks of age. This virus often is found in mixed infections with ETEC and cryptosporidia.

(2) Clinical findings. Clinical findings in pure rotavirus infections are diarrhea, dehydration, and anorexia. The condition may last for a few days, and recovery is usually uneventful. Combined infections with other pathogens (e.g., *E. coli*) result in a clinical picture of undifferentiated neonatal diarrhea indistinguishable from ETEC or combined enteric pathogens.

(3) Etiology and pathogenesis

(a) The condition is caused by one of several strains of RNA rotavirus. The virus attacks absorptive cells at the tips of the villi of the small intestine. Loss of these mature epithelial cells results in malabsorption (lactase washout), osmotic diarrhea, dehydration, electrolyte loss, and acidosis.

(b) Intestinal regeneration and epithelial cell function return to normal within approximately 7 days, although normal growth rates for the calf may take 10–21 days to return.

(4) Diagnostic plan. Accurate diagnosis depends on laboratory confirmation.

(5) **Laboratory** tests. The virus may be isolated from fresh feces or intestine. Tests that should be performed include electronmicroscopy, immunofluorescence, latex agglutination, and ELISA.

(6) Therapeutic plan. The treatment is as for ETEC or undifferentiated neonatal diarrhea [see II B 3 a (6)].

(7) Prevention

(a) Management strategies. The principles of control are the same as for undifferentiated neonatal diarrhea — limit exposure to the organism, ensure colostral intake, and increase specific antibody levels by vaccination of the calf or dam.

(b) Vaccination of calves has given less than satisfactory results in field studies. For vaccination of the dam to provide protection to the calf, it must occur at a time of colostrum production, and continued feeding of milk or colostrum with high antibody titers during the times calves are susceptible to infection is necessary. This may require management changes in veal or dairy operations.

c. Coronavirus diarrhea

(1) Patient profile. Coronavirus causes diarrhea in beef or dairy calves under a variety of management practices. The age range of infected calves is generally 5–20 days.

(2) Clinical findings. Clinical findings are similar to other cases of neonatal diarrhea, with the exception that flecks of frank blood may be seen in the feces of coronavirus-infected calves.

(3) Etiology and pathogenesis. Coronavirus replicates in and damages the villus epithelium of both the small and large intestines. The crypt cells also are damaged, which results in a longer rejuvenation time for cell repair and replacement. Loss of epithelial cells results in malabsorption, maldigestion, and diarrhea.

(4) Diagnostic plan. Diagnosis is based on confirmatory laboratory tests as in rotavirus infection [see II B 3 b (4), (5)].

(5) Therapeutic plan and prevention. Therapy and prevention is as for rotavirus infection, with the added caveat that calves with coronavirus diarrhea take longer to recover because of crypt cell destruction. Convalescence may be prolonged and weight loss significant. Attention to nutritional supplementation may be necessary.

d. Cryptosporidiosis diarrhea

(1) Patient profile and history. Neonatal dairy or beef calves are affected.

(2) Clinical findings

(a) The clinical signs are indistinguishable from other causes of neonatal diarrhea. Calves are usually 1–3 weeks of age. Tenesmus may be a feature of the condition as is weight loss and the persistence of the diarrhea.

(b) Appearance. Severe dehydration, weakness, and recumbency are not characteristic of uncomplicated cases of cryptosporidiosis.

(3) Etiology and pathogenesis

(a) The causative agent is the protozoan parasite, ***Cryptosporidium parvum.*** Infective oocysts are ingested and develop through six stages within the lower small intestine, large intestine, and cecum. The parasite produces villus atrophy, impairment of digestion, and absorption with a resultant mild diarrhea in uncomplicated cases. The organism is frequently seen in combination with other agents that cause neonatal diarrhea. This increases the severity of the diarrhea and the clinical effect on the calf.

(b) The prepatent **period** is 2–7 days, and sporulated oocysts may be passed for 3–12 days in the feces.

(4) Diagnostic plan. Diagnosis depends on laboratory confirmation.

(5) **Laboratory** tests. Fecal oocysts may be detected by fecal floatation, direct staining of fecal smears, immunofluorescence, or ELISA.

(6) Therapeutic plan

(a) Medical therapy. Diarrhea with uncomplicated cases of cryptosporidiosis is self-limiting. However, there is no recommended or specific treatment for cryptosporidiosis. On an experimental basis, the anticoccidial agent **halofuginone** has been used with some success at 60–125 μg/kg orally for 7 days.

(b) **Supportive** therapy. Other treatments are supportive and similar to treatments of all diarrheas (e.g., warmth, oral or intravenous fluids, milk feeding in small quantities several times daily, oral protectants).

(7) Prevention

(a) The only control at present is to limit exposure of calves to the organism. Procedures used include:

(i) Segregation of infective calves

(ii) Separation of feeding utensils

(iii) Manure removal

(iv) Disinfection of the environment with 5% ammonia or chlorine–dioxide-based disinfectants

(b) Clients should be warned that *C. parvum* is a **zoonotic** agent and causes diarrhea in humans. The condition has serious implications in immunocompromised individuals.

e. **Giardiasis**

(1) Patient history and etiology. *Giardia* (e.g., *G. duodenalis*) has been recovered in feces of diarrheic calves. Its etiological significance is yet to be proven because infection occurs experimentally, but clinical signs do not develop.

(2) Clinical findings and therapeutic plan. A syndrome is described of *Giardia*-associated, chronic, pasty diarrhea lasting for 2–6 weeks. Growth is depressed. Fenbendazole at 11 mg/kg has shown efficacy against this parasite.

(3) *Giardia* species cause disease in humans, so a **zoonotic** potential exists when calves are excreting the organism.

f. **Salmonellosis**

(1) Patient profile and **history.** Salmonella usually affects calves older than 10–14 days of age.

(2) Clinical findings. **Three** syndromes have been described in calves 10 days to 3 months of age. These syndromes are:

(a) Peracute — sudden death, neurological signs (opisthotonos, convulsions), or gastrointestinal signs (abdominal pain, diarrhea).

(b) Acute—fever, anorexia, depression, diarrhea, and dehydration. Diarrhea progresses from watery to mucus/epithelial casts to hemorrhagic feces.
(c) Chronic—loose feces with poor growth rates and ill-thrift

(3) Etiology and pathogenesis. Salmonella ***dublin*** and ***S. typhimurium*** are the most common *Salmonella* isolates. The pathogenesis of the diarrhea is similar to that described for salmonellosis in the adult [see II B 1 d (3)]. A bacteremic form also may exist.
(4) Diagnostic plan. Diagnosis is supported by laboratory findings and farm history.
(5) Laboratory tests. Necropsy and culture findings of feces or intestinal contents confirm the diagnosis. Repeated culture attempts may be necessary to isolate the organism from feces.
(6) Therapeutic plan and prevention. Therapy and prevention are as for the adult [see II B 1 d (7), (8)].

g. **Clostridial** diarrhea
(1) Patient profile and history. This type of diarrhea may be reported as an outbreak in rapidly growing, vigorous, nursing calves. It is more commonly reported as a disease of older calves or sheep.
(2) Clinical findings. Sudden death may be the most common presentation. Less acute disease may present as abdominal pain, distention, and hemorrhagic enteritis.
(3) Etiology and pathogenesis
(a) ***Clostridium*** perfringens normally is found in the intestinal tract and may proliferate at times of abrupt feed changes or overfeeding of carbohydrates. Enterotoxins (alpha, beta, epsilon, and iota) produced by the various organism types produce the clinical signs.
(b) C. perfringens type A produces a hemorrhagic enteritis in Europe.
(c) C. ***perfringens*** type B causes diarrhea in many types of neonates in Europe, South Africa, and the Middle East.
(d) C. ***perfringens*** type C causes hemorrhagic enteritis of calves in North America.
(e) C. ***perfringens*** type E causes necrotic hemorrhagic enteritis but is rare.
(4) Diagnostic plan. A tentative diagnosis may be made based on subjective and clinical findings but requires laboratory confirmation.
(5) Laboratory tests. Necropsy and/or mouse inoculation with intestinal contents and neutralization studies with specific antitoxin confirm the diagnosis.
(6) Therapeutic plan. Therapy with antibiotics (penicillin) and hydration support may be attempted but are rarely successful because of the peracute nature of the disease. Similarly, specific antitoxin therapy is rarely available.
(7) Prevention. Active immunization of the dam may be employed using toxoids appropriate for the serotype. Vaccination of the dam before parturition (6–8 weeks) imparts protection to the calf in the colostrum.

h. Dietary causes of diarrhea
(1) Patient profile
(a) Amounts of liquid feed. Dairy calves should be fed fresh colostrum at the rate of 10% body weight (BW) per day for the first 3 days. This should be divided into two or three feedings. Following this, calves may be fed milk or milk replacer at 8%–10% BW/day not to exceed 2 L/feeding. Overfeeding in total or at any given feeding may result in diarrhea. In a cold climate, the total amount of milk fed may need to be increased to 12%–14% BW/day.
(b) Type of liquid feed. Satisfactory performance can be achieved with the use of colostrum, whole milk or milk-based milk replacers.
(i) Colostrum may be collected from mature cows and stored by freezing, refrigeration, fermentation, or through the addition of chemical preservative. Colostrum is diluted 2:1 or 3:1 with water before feeding.
(ii) Milk replacers should be milk-based as plant proteins and starches are not well digested by the neonatal calf. Maldigestion of these ingredients results in poor growth rates and diarrhea. Milk replacers should also contain 10%–20% fat, which is an energy source and limits diarrhea.
(c) Method of feeding. Feeding via nipple feeder or open bucket in neonates causes no significant differences in health or growth rates. An exception to this is veal calves described as "rumen drinkers" where older calves (5–6 weeks) ingest milk from buckets directly into the rumen. This results in recurrent ruminal tympany/fluid distention, inappetence, poor growth rates, and the production of clay-like feces.
(2) Clinical findings and therapeutic plan. The clinical signs are caused by ruminal hyperkeratosis and intestinal villous atrophy. Treatment includes conversion to nipple pail nursing or weaning onto roughage diets.

i. Infectious bovine rhinotracheitis may cause diarrhea in young calves as part of a systemic infection (see Chapter 6 II B 6 a).
j. Bovine viral diarrhea is discussed in II B 1 a.
k. Prolonged antibiotic therapy. Prolonged oral antibiotic therapy may predispose calves to intestinal overgrowth of pseudomonas, proteus, or fungi. Intractable diarrhea may result.

STUDY QUESTIONS

DIRECTIONS: Each of the numbered items or incomplete statements in this section is followed by answers or by completions of the statement. Select the ONE numbered answer or completion that is BEST in each case.

1. What conditions should be included on a list of differential diagnoses for diarrhea in a calf younger than 1 week of age?

(1) Enterotoxigenic *Escherichia coli* (ETEC) diarrhea, coronavirus diarrhea, rotavirus diarrhea
(2) Salmonellosis, clostridiosis, primary disaccharidase deficiency
(3) Primary disaccharidase deficiency, rotavirus diarrhea, ETEC diarrhea
(4) Coronavirus diarrhea, salmonellosis, coccidiosis
(5) Giardiasis, transmissible gastroenteritis (TGE), clostridiosis

2. Which one of the following statements regarding traumatic reticuloperitonitis (TRP) is correct?

(1) Therapy should include antibiotics and confinement
(2) TRP is least common in mature dairy cows.
(3) Left paramedian abdominocentesis should be attempted to diagnose peritonitis.
(4) Affected animals go off feed abruptly but continue to produce milk at 75% of the normal production level.
(5) Affected animals often stand with their hind feet elevated to relieve the acute pain.

3. A veterinarian is called to see a 5-year-old jersey cow. She has been fresh for 2 months and was treated for hardware disease by the owner shortly after calving. She is now milking at a level that is 50% lower than would be anticipated and is selectively inappetent, preferring roughage to her concentrate ration. The owner has been treating her with rumenatorics for 2 weeks, but has seen no response to therapy. Physical examination reveals a distended abdomen with mild gaseous distention of the left paralumbar fossa and succussible fluid in the lower right abdominal quadrant. The cow is passing scant, pasty feces and appears to be thin and mildly dehydrated. The cow exhibits four weak rumen rolls per minute but is not chewing her cud. Her heart rate is 30 bpm. The most probable diagnosis is:

(1) chronic bovine virus diarrhea (BVD).
(2) simple indigestion.
(3) acute localized peritonitis.
(4) vagal indigestion.
(5) Johne's disease.

Questions 4–5

In January, a veterinarian is called to evaluate a herd of Holstein cows that are housed indoors. Eighty percent of the milking cows have developed acute, profuse, diarrhea over the last 12–24 hours. Occasionally, the feces are bloody and mucoid. The milk production in the herd has decreased by 50%. The cows are fed an 18% protein dairy ration and alfalfa-timothy hay. A new source of grain was introduced in the last 3 days. Other animals (virgin heifers and bulls) are normal, although they are fed a similar diet. Physical examination reveals that some of the animals are mildly dehydrated. The vital signs are normal and no other abnormalities are detected. Complete blood cell counts (CBCs) and biochemisiry profiles obtained from several clinically affected cows are essentially normal except for increases in the packed cell volume (PCV) and total protein (TP).

4. What is the most likely diagnosis?

(1) Bovine virus diarrhea (BVD)
(2) Salmonellosis
(3) Winter dysentery
(4) Arsenic poisoning
(5) Rotavirus diarrhea

5. What is the appropriate next step?

(1) Treat all affected animals with potentiated sulfonamides, flunixin meglumine, and oral or intravenous fluids.
(2) Treat all affected animals with broad-spectrum antibiotics and intestinal protectants.
(3) Treat all affected animals with oral charcoal and systemic British antilewisite (BAL).
(4) Run serologic tests to confirm the diagnosis.
(5) Wait for the disease to run its course. Supportive care (e.g., fluid therapy and astringents) is indicated for dehydrated animals.

DIRECTIONS: Each of the numbered items or incomplete statements in this section is negatively phrased, as indicated by a capitalized word such as NOT, LEAST, or EXCEPT. Select the ONE numbered answer or completion that is BEST in each case.

6. All of the following are true statements regarding grain overload in sheep EXCEPT:

(1) administration of oral magnesium oxide to sheep not showing signs of shock and dehydration is one correct form of therapy.
(2) absorption of both D- and L-lactate occurs, but only the D-lactate causes acidemia.
(3) this condition occurs when gram-positive rods overgrow normal rumen flora.
(4) emergency surgery will allow salvage of animals that are convulsing and in shock.
(5) the severity of clinical signs depends on the amount and particle size of the ingested grain.

7. Which one of the following statements is NOT correct regarding calves born to cows 3–9 months after an episode of bovine virus diarrhea (BVD) in a nonimmune population?

(1) The calves may be born with mucosal disease that is unresponsive to therapy.
(2) The calves may be born with cerebellar disease.
(3) The calves are immunotolerant to the BVD virus but they are incapable of mounting an antibody response to it.
(4) The calves are chronic shedders of the BVD virus.
(5) The calves may appear normal or suffer from ill thrift.

8. All of the following statements concerning bovine malignant catarrh (BMC) are true EXCEPT:

(1) BMC occurs sporadically and is usually spread from cow to cow.
(2) BMC causes a vasculitis and atypical proliferation of lymphocytes.
(3) BMC causes lymph node enlargement.
(4) BMC causes a panophthalmitis. Corneal opacity usually begins peripherally and spreads centrally.
(5) BMC is frequently accompanied by nervous system signs.

9. All of the following are suspected pathophysiologies of left displacement of the abomasum (LDA) EXCEPT:

(1) reduction in abomasal motility due to hypocalcemia.
(2) reduction in abomasal tone through stabling and lack of exercise.
(3) reduction in abomasal motility from toxins released during concurrent disease.
(4) reduction in abomasal motility due to a decrease in volatile fatty acid (VFA) production.
(5) reduction in abomasal motility due to an increased dietary concentrate:roughage ratio.

ANSWERS AND EXPLANATIONS

1. The answer is 1 [II B 3]. Included on a list of differential diagnoses for diarrhea in a calf younger than 1 week of age would be enterotoxigenic Escherichia *coli* (ETEC) diarrhea, coronavirus diarrhea, and rotavirus diarrhea. Salmonellosis, clostridiosis, and coccidiosis are usually a cause of diarrhea in older calves. Primary disaccharidase deficiency has not been reported in calves. *Giardia lamblia,* although recoverable from the feces of some calves with diarrhea, is unproven as a cause of neonatal calf diarrhea. Transmissible gastroenteritis (TGE) is a disease of swine.

2. The answer is 1 [I E 1]. Traumatic reticuloperitonitis (TRP, hardware disease, traumatic gastritis, traumatic reticulitis) is most common in mature dairy cows. Clinical signs include an abrupt drop in milk production (to less than 50% of normal) and odd postures (e.g., the animal may stand with its hind feet in a gutter in an attempt to relieve diaphragmatic reticular pressure). A right, not a left, paramedian approach is recommended to collect peritoneal. fluid for laboratory analysis. Conservative therapy entails confinement and administration of antibiotics; rumenotomy may be necessary if conservative management fails and economics warrant surgical intervention.

3. The answer is 4 [I A 3]. The clinical findings suggest vagal indigestion. The history may include mild but repeated bouts of transient indigestion with signs of anorexia, decreased milk production, mild bloat, weight loss, abdominal distention, and decreased amounts of manure. The animal may have experienced an episode of traumatic reticuloperitonitis (TRP) in the past. Chronic bovine virus diarrhea (BVD) is seen in young animals with diarrhea, a stunted growth pattern, and lameness. Simple indigestion resolves within a few days. Acute local peritonitis (while perhaps the initial cause of this chronic disease) would have resolved. Intractable diarrhea is the most evident complaint and finding in animals with Johne's disease.

4–5. The answers are: 4-3, 5-5 [II B 1 el. The most likely diagnosis is winter dysentery. The subjective findings (e.g., housed cattle, month) support a diagnosis of winter dysentery. The cows present as essentially normal, except for an explosive outbreak of diarrhea. These findings eliminate salmonellosis and arsenic poisoning from the list of differential diagnoses. Rotavirus has not been demonstrated as an etiologic agent for diarrhea in adult ruminants. A virulent virus serotype responsible for bovine virus diarrhea (BVD) would cause similar, but more severe, clinical findings (e.g., severe dehydration).

A bovine coronavirus is the etiologic agent of winter dysentery. Treatment consists of supportive care and waiting for the disease to run its course. Antibiotics are unnecessary and ineffective. British antilewisite (BAL) is a treatment for arsenic toxicity. Serologic studies would be of no value for diagnosis.

6. The answer is 4 [I A 2]. In a convulsing animal in shock as a result of grain overload, surgery is not economically warranted because these animals have a poor prognosis for recovery. Lactic acidosis resulting from grain overload occurs when gram-positive rods overgrow the normal rumen flora. Absorption of both D- and L-lactate occurs, but only D-lactate causes acidemia. The severity of the clinical signs depends on the amount and particle size of the grain ingested; finely ground feeds are associated with more severe clinical signs. Administration of oral magnesium oxide to sheep not showing signs of shock and dehydration is one appropriate therapy.

7. The answer is 1 [II B 2 b]. Calves that have been exposed to the bovine virus diarrhea (BVD) virus in utero are not born with mucosal disease; rather, they may develop a fatal mucosal disease sometime later after being exposed to a cytopathic form of the virus. Affected calves are immunotolerant to the virus but are unable to mount a humoral antibody response against it. Affected calves may appear normal at birth, or they may suffer from ill thrift. Affected calves are chronic shedders of the BVD virus.

8. The answer is 1 [II B 1 b]. Bovine malignant catarrh (BMC) occurs sporadically and is caused by a wildebeest-associated virus (in Africa) or a sheep-associated virus (in North America). The virus is not known to be spread cow-to-cow. Disease outbreaks are often associated with ewes lambing near cattle. BMC causes vasculitis, atypical proliferation of lymphocytes, panophthalmitis, and lymph node enlargement and is frequently accompanied by nervous system signs.

9. The answer is 4 [I C 1]. High dietary concentrate: roughage ratios increase the production of volatile fatty acids (VFAs). VFAs are known to decrease intestinal motility; it is hypothesized that this leads to gas build-up within the abomasum and eventual displacement of the left abomasum. It is thought that abomasal tone is also negatively affected by low serum calcium levels, circulating toxins, and lack of exercise in deep-bodied cows.